The Biliary Tract

With special reference to the common bile duct

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To Elsie and Ralph

PREFACE

This monograph has been prepared to correlate clinical features laboratory investigations operative procedure and biliary tract functions for the benefit of the gastroenterologist the general practitioner, the surgeon and the investigator

Achievements and errors have been recorded Facts and theories have been presented Most of all specific methods for the treatment of biliary tract disorders are made available so that technical mastery going hand in hand with clinical management should permit early and complete relief of symptoms without the necessity for multiple operations And in some cases without operation

Clinical and radiologic methods for diagnosis reveal correctible diseases of the bile ducts and gallbladder in more individuals each year It behooves the physician and surgeon to relieve these abnormalities whether they be benign or malignant inflammatory or anomalous primary or secondary Cholecystitis and cholelithiasis should be remediable within the limits of human error

Whipple and Ravdin have led the way in technical and physiological advances which prepare the patient to undergo the most radical corrective measures Dardinski Mirizzi Boyden Michels Popper and Wilkie have given to us the basic facts concerning the ducts which our present generation of surgeons utilizes at every operation Deaver Kehr Moynihan Lahey Cattell the Mayos Thorek Estes Pribram Colp Mulholland Doubillet Brunschwig Mallet Guy and hundreds of students whose work is tabulated in the bibliographies have contributed the experiences which today permit safe adequate and frequently curative correction of invalidism due to the hepato-pancreato-duodenal biliary tracts Einhorn Lyons Bockus Lichtman Weiss Hanger Graham Lucke Schiff Tumen and other internationally famed gastroenterologists and medical scientists have done more than publications alone may recount

It is to these men and their colleagues in the laboratories that this monograph is respectfully dedicated Their art ideals knowledge curiosity imagination honesty and industry have been expressed in the benefits reaped by their happy patients

The author wishes to acknowledge his gratitude to Dr I S Ravdin for his maintained integrating interest to Dr O V Batson for his apt and unfailing scientific altruism and to Dr R Goldsmith for his patient criticisms Appreciation is also expressed to Dr W E Ehrich Dr H R Hawthorne Dr S Lorber and Dr H J Tumen for their advice

The chapter on radiology was guided by suggestions from and prepared in collaboration with Drs M Hermel J Gershon Cohen and R Katzen Dr I Young's critical review of the chapters on pathology and the pancreas was succinct and invaluable The author acknowledges with gratitude the preparation of the chapter concerning anesthesia for biliary tract surgery by Dr L E Fredericks

The grand cooperation by editors of various medical journals in their authorization for use of cuts and illustrations is greatly appreciated The author thanks the editors of *American Journal of Gastroenterology*, *American Journal of Pathology*, *American Journal of Radiology*, *Annals of Surgery*, *Archives of Surgery*, and *Surgery, Gynecology and Obstetrics* for their favors I am particularly grateful to the Charles Pfizer Co and their medical Department for the use of several four color plates which have appeared in the Pfizer Spectrum of the J A M A

The typists, artists, bibliographers and editorial assistants have, by their many cooperative kindnesses, engraved a lasting protection within my myocardium against spasm and sclerosis Thank you, all

JULIAN A STERLING

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INTRODUCTION

A General

Biliousness is part of the folk lore in the earliest medical writings The ancients were familiar with jaundice, with colic and with the irritable cranky and mean disposition associated with disturbances in the "bile" humors Older medical teachings recognized the beneficial results of calomel purgatives and emetics in relieving biliary tract disease

In addition the soothsayers who examined the sacrificial livers for prophecies, identified the structure and ducts of the liver in the herbivorous species Comparative anatomy and human physiology however were partly helpless because the sacrificed animals had not been afflicted with gallstones

The first recognition of gallstones was simultaneous with the discovery of America At that time Paracelsus recorded their presence in cadavers he also presented the theory that stones were produced around impurities in the bile

Statistics indicate that over 10 per cent of adults have chronic cholecystitis and that up to 30 per cent of women over 40 years of age have gallstones Withal however, the gallbladder and bile ducts are probably incriminated too frequently as the source of indigestion

On the other hand biliary tract disease may be asymptomatic On many occasions autopsy examination reveals unsuspected gallstones Coexistent disease of the biliary ducts does not necessarily cause death But inflammation and dilatation of the ducts in the presence of obstructive jaundice and biliary cirrhosis or other degenerative phenomena can be sufficient to disturb homeostasis There is no doubt that death can be primarily produced by cholangitis pyelophlebitis liver abscess hepatorenal failure and obstructive jaundice when due to bile duct calculi or tumor

Remedial therapy to the biliary tract has achieved clear maturity during the past fifty years Differential diagnosis of jaundice, the use of oral cholecystography and other adjuvant radiologic techniques careful anatomical studies and the alteration of surgical approach from palliation to resection have all given new vistas to diseases of the bile ducts Many problems still exist the exact origin of calculi the nature of pancreatitis differential diagnosis of certain phases of intrahepatic jaundice the efficacy

of resection for malignancy and the effects of denervation procedures have not been entirely clarified

We have learned to recognize the indications for surgical therapy in jaundice, methods for the preoperative preparation and selection of anesthesia in the older and poor risk patients with damaged livers the use of radiography before, during and after bile duct operations, the procedures for providing a patent papilla and the modes for repairing congenital and traumatic strictures We have made much progress in the resection of biliary tract malignancy We have learned much since Dr Glisson described the papilla of Vater (before Vater) in 1690 and since Laurentius Heister reported the "beautiful and surprising valves of a spiral figure which divided the cystic duct into a number of cells "

B The Bile Ducts (Fig 1)

The bile duct conduit transports bile from the liver to the duodenum This pipeline includes a 'surge chamber' (represented by the gallbladder)



FIG 1 CHOLECYSTOCHOLANGIOGRAM

Operative cholecysto cholangiogram under spinal anesthesia Contours relationships size and position of the bile ducts and the gallbladder are visualized in the normal individual

permitting both storage and availability The duct also has a "nozzle" at its termination which increases the flow of bile to jet stream velocity These concepts conform to hydraulic principles

The common bile duct has a constantly curving course Its termination at the papilla of Vater is like a funnel in that the diameter of the lumen decreases rapidly

The external appearance of the terminal (transduodenal) segment of the common bile duct is that of an expanded bulb This enlargement occurs where the pancreatic duct appears to join the termination of the common bile duct This bulbous character for many years led to a belief that a space was formed by the confluence of the pancreatic and common bile ducts

When the enlarged segment is bisected it is found that it is a mass of sphincter (of Oddi) muscle The diameter of the common bile duct lumen is very narrow There is then no space (ampulla) but actually a mass (papilla) present at the transduodenal termination of the common bile duct

There are other differences between the extraduodenal and transduodenal (transpapillary) common bile duct The extraduodenal duct is translucent, thin walled (0 5 to 1 5 mm) and flexible and contains three to four gland orifices per square centimeter The papillary portion of the duct is dull thick (0 3 to 1 9 cm) rigid and has numerous folds or mucosal reduplications acting like valvules

The pancreatic and common bile ducts generally maintain their identity through the duodenal wall Careful examination of intact cleared specimens after filling the ducts with opaque dyes demonstrates that in 80 per cent of specimens the duct terminations are absolutely separated or there is a shallow common channel for 1 or 2 mm In 14 per cent of cases the common channel for pancreatic and common bile ducts is in the distal one third of the papilla In only 6 per cent does the common channel for the ducts traverse more than half of the papilla

Consideration of the disposition and character of the papillary sphincter indicates that the presence of interductal reflux depends upon the existence of a common channel for the bile and pancreatic ducts for a distance greater than half the length of the papilla Such conditions exist in less than 20 per cent

The percentage of interductal reflux (20 per cent) has been confirmed by studies of postoperative cholangiograms It is important to relate the site of normal interductal reflux to the site of stone impaction Usually the calculus is lodged at the distal end of the extraduodenal common bile duct It is unable to enter the duodenum through the normal common bile duct unless it is less than 0 3 cm in diameter

Because of the mass of tissue present at the papilla a stone within the

common bile duct propelled by increasing intracholedochal pressure seeks areas of least resistance These may be toward the pancreas, its duct or the duodenal wall Frequently, an abnormal interductal reflux is established through such traumatic or inflammatory choledochal pancreatic fistulae Pancreatitis is present in these cases

Flow of bile through the papilla can occur at pressures of from 9 0 to 23 0 cm of bile, sphincter resistance normally is 12 0 to 15 0 cm of bile These are altered by abnormalities at the termination of the extraduodenal common bile duct and within the papilla The papilla is from 8 0 to 12 0 cm from the pylorus It is above or on a level with duodenal mucosa in about 75 per cent of persons It is 0 3 to 1 9 cm wide and 0 7 to 1 9 cm long While the diameter of the normal extraduodenal common bile duct is 0 7 cm , the diameter of the orifice through the papilla is 0 1 cm

Abnormalities of the papilla are reflected by changes in the extraduodenal common bile duct The most common change is that of dilatation to the intrahepatic ducts, the common hepatic duct and the common bile duct The common bile duct is abnormal when greater than 1 cm in diameter This can be seen in patients with stricture, spasm or tumor

Vater's description (1748) of the common bile duct termination was amplified by Oddi (1887) Many investigators have since reviewed the structure of the distal end of the common bile duct, but it was Robson particularly who influenced clinicians through his descriptions of the four major types of junction of pancreatic and common bile ducts However, there is some confusion regarding the anatomical findings of the distal segments of the common bile and pancreatic ducts

An example of inconsistency is that the terms "papilla" and "ampulla" are interchanged Etymologic analysis indicates that an "elevation or swelling' (the papilla) such as a nipple should not be confused with a dilated end of a vessel, canal, or duct' (the ampulla), particularly when the former means a mass, and the latter a 'space " Hence the so-called 'carcinoma of the ampulla" (or tumor of a space) is a misnomer

It is true that an 'ampulla has been described in the human embryo wherein the hepatic pancreatic duct joins the fused duodenum during the solidification phase but it consequently regresses (fig 2) This embryologic "ampulla" is not found in the adult bile duct All that normally remains is a small depression in the mucosal septum between the termination of the common bile and pancreatic ducts

The common bile duct on intubation, by radiographs and at the operating table differs from that seen on post mortem examination The living physiologic pathology often is difficult to evaluate The clarification of a confusing picture at operation depends upon the several findings of inspection, palpation, injection and radiographs Careful confirmatory ob

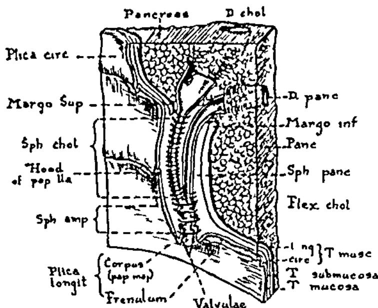


FIG 2 PAPILLA BOYDEN S CONCEPT

Schematic reconstruction of the papillary termination for common bile duct (D chol) and pancreatic duct (D panc) within the duodenal lumen Boyden indicates a sphincter for the common channel (Sph Amp) incorrectly identified as an am pulla (Courtesy of Dr Boyden and *Am J Anat*)

servations are often required to establish the nature of and the diagnosis of a common bile duct abnormality In addition other disease may camou flage may misrepresent or may be identical with bile duct disease

C Definitions

The terms which are used in this text are defined as follows

1 AMPULLA OF VATER a dilated common channel at the termination of the common bile and pancreatic ducts a dilatation in the lumen of the common bile duct at its distal termination

2 CARUNCLE the same as the 'major duodenal papilla" (vide infra)

3 COMMON CHANNEL the course of the lumen in the papilla formed distal to the union of the common bile and pancreatic ducts

4 CYSTIC PEDICLE cystic duct between gallbladder and the common bile duct surrounded by reflections of the hepato-duodenal ligament including adjacent veins, artery and nerves extending from the hepatic pedicle to ramify over the gallbladder

5 HEPATIC PEDICLE between the pyloroduodenal region and the liver hilum surrounded by expansions of the hepatogastric and hepatoduodenal ligament representing the right lateral portion of the lesser omentum and

the superior border of the foramen of Winslow, which contains the common and hepatic bile ducts, the hepatic artery and its branches, the portal vein and many nerve plexuses.

6. MAJOR DUODENAL PAPILLA: the swelling of the terminal portion of the common bile and pancreatic ducts, usually beginning at the entrance of the ducts into the duodenum and terminating within the duodenal lumen upon the summit of which are the duct orifices.

7. MINOR DUODENAL PAPILLA: firm elevation of the duodenal mucosa frequently present at the orifice of the accessory pancreatic duct. This is proximal to the major duodenal papilla.

8. ORIFICE: the ending of the lumen of the distal termination of the pancreatic and/or common bile ducts, visualized as a space or a hole in the duodenal mucous membrane.

9. PAPILLA (AND "PAPILLA OF VATER"): same as "major duodenal papilla" (vide supra).

D. Bile and Calculi

The true nature and function of bile was first identified by Sydenham in 1669. It was at this time that jaundice was recognized as a symptom rather than as a disease. The identity of bile had been recognized by its color, odor and taste (in vomitus) before this. Castiglione states that Regnier de Graaf, in the seventeenth century, had studied the bile from an external traumatic fistula.

In 1723 Laurentius Heister stated that gallbladder bile was more viscid than liver bile. MacLurg (1772) described gallbladder bile carefully and also differentiated it from hepatic bile. It was actually in the last 20 years of the nineteenth century that chemical examinations were done (Maly, Brand) to determine the character of the total solids present in bile. Rous and McMaster initiated studies which Ravdin, Johnston, Reigel and Morrison carried to a clear cut conclusion to indicate the exact function of the gallbladder with relation to the bile (cf. Chapter 3).

In 1863 Thudicum stated that biliary calculi resulted from a decomposition of the bile, the process resembling putrefaction. Since then there has been presented the "stasis infection" theory of Naunyn, the "stasis hypercholesterolemia" theory of Aschoff and Baemeister, the "physico-chemical" theory promulgated by Sterling and confirmed by Martensson and Olin (cf. Chapter 4).

The stumbling block to all theories concerning stone formation is that there has been no reliable method for determining differences between bile obtained from calculous or from non calculous gallbladders. Newer studies in progress with relation to the electrophoresis patterns, may well provide the key clue.

Chemical electrophoresis patterns may also provide the clue regarding carcinoma The interrelationships between stones and malignancy is most intriguing

E Therapy

Successful surgical techniques for benign disease of the biliary tract have been mastered by surgeons who were conscious of the anatomical obstacles and physiological responsibilities These procedures have rapidly embraced resection for malignancy which obstructs the bile ducts

Winiwarter did the first cholecysto-anastomosis in 1880 in Austria This surgeon did a cholecystocolic anastomosis Around 1911 or 1912 Kousch in Berlin had two temporary successes in removal of the pancreas with re implantation of the pancreatic duct into the jejunum

Among surgeons during the nineteenth century who operated on the pancreas were Becourt Bigsby da Costa and Jeune Halsted and Mayo reported successful results in resection of papillary carcinoma However it was not until 1930 or 1940 that physiologic chemistry (vitamin K) anesthesia (endo-tracheal tube) and technique (Whipple Parsons Brunschwig Cattell Lahey Walton Graham and Gordon Taylor) permitted consideration of elective resection of carcinoma involving the pancreas and bile ducts Clinical and radiologic diagnosis has kept pace with other developments Differential clarification of jaundice by laboratory and clinical methods (Hanger Bockus Popper and others) has increased early recognition of obstructive jaundice Careful radiology (inverted 3 and pad signs) now aids the earlier identification of certain tumors

F Prognosis

From Wirsung and Glisson through Vater Santorini and Oddi and to the present the common bile duct has not changed However there has been an impediment to the constant surge of progress

In the early years of the twentieth century based upon certain incomplete studies the concept of an ampulla crept into clinical and from there into other writings This concept has delayed further progress and has been responsible for much theory

Physicians as well as other scientists frequently re evaluate facts and when indicated take proper steps to reorganize procedures Often the impetus to appraisal may be absent because of the ease and comfort accompanying lines of least resistance

However facts may be misinterpreted because of inadequate terminology Such is the case in the poor usage applied to the terminations for the common bile and pancreatic ducts Perhaps a careful examination of the facts will be valuable

For at least 50 years the duodenal termination of the pancreatic and the common bile ducts have been identified as an "ampulla." The term suggests the presence of a "space" or a dilatation, presumably a confluence for the ducts which merge to form a wide lake. This is fallacious.

The "papilla of Vater" is a "nipple" or mass of fibrous and muscular tissue carrying within it very tiny and narrow ducts. This is a known fact (Dardinski, Boyden, Sterling).

The papilla may be elevated above, equal to or depressed below adjacent duodenal mucosa but its position does not eliminate the fact of its mass.

The term "ampulla" is defended by some as indicating that portion of the terminations of the ducts other than the mound or eminence which is visible or palpable from within the duodenum. Yet these reports will also acknowledge the presence of sphincter muscle within the same areas. The sphincter muscle, whether described as a unit or in three or more parts, is the mass comprising the papilla. The sphincter of Oddi surrounds the terminations of the common bile and pancreatic ducts and within the papilla it is most unusual for an ampullary dilatation to be present.

It has been determined that an ampulla may be normally present in about 5 per cent of normal individuals. This percentage is included in the individuals who are known to have a common channel for both bile and pancreatic ducts. The rarity of the actual "ampulla" presents a serious contrast between nomenclature and fact.

In addition, it is not conceivable that "tumor," "fibrosis" or "inflammation" could properly apply to a "space" or "ampulla." If used, this could apply only to such areas of the extraduodenal common bile or pancreatic ducts which have become abnormally enlarged.

The concept of a space or an ampulla infers that there is a constant interductal reflux. This is a misconception which is based on the fallacious terminology. It may continue to provoke new and misdirected therapy. The facts should all be apprised in full, before theories are proposed. Therapy of pancreatitis, for example, would then no longer require a large charge of buck shot from a double barrelled shotgun.

Many more misconceptions may result from incorrect and inadequate terminology. It is strongly urged, therefore, that among other things the designation given to the terminations for the common bile and pancreatic ducts no longer be anything but a "papilla."

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2

ANATOMY

Gallia est omnis divisa in partes tres, " The biliary ducts have three major anatomical divisions (A) intrahepatic, (B) extraduodenal and (C) trans-duodenal (fig 3)

A Liver and Intrahepatic Bile Ducts

1 Liver

The basis for all gall is the liver This huge gland averages 1500 grams in weight, occupies approximately 2500 cc beneath the right leaf of the diaphragm and is shaped like a thick béret It moves with the diaphragm, can be displaced downward by the surgeon's hand and rotated up to 45 degrees *around an axis formed by the portal vein (inferiorly) and hepatic* veins (superiorly)

Arborization of the portal vein divides the liver into right and left lobes The main (central) fissure is generally to the left of the liver at about 35 degrees *from the sagittal plane The left, smaller lobe is divided by* extensions of the falciform ligament into medial and lateral segments The medial segment of the left lobe contains the quadrate and the Spigelian lobes The right lobe is comprised of dorsocaudal, intermediate and ventrocranial segments

Although it appears to be without visible means for support, there are peritoneal folds and connective tissues around the vascular pedicles which suspend the liver in mid torso There are seven named ligaments which surround it These are 1) falciform, 2) coronary 3) right triangular (lateral) 4) left triangular (lateral), 5) round (lig teres) 6) hepatogastric and 7) hepatoduodenal

1 The falciform ligament is composed of reduplicated peritoneal folds extending between the left lobe of the liver and the adjacent diaphragm and right rectus muscle The round ligament is given attachment at the antero-inferior liver margin where the falciform ligament joins it

2 The coronary ligament surrounds the hepatic veins at the dome of the liver and suspends it to the under surface of the right diaphragm A portion of this ligament merges posteriorly with the *capsule of the right* kidney

3 The right triangular ligament extends between the upper surface of

the liver and the right diaphragm at the lateral aspect of the coronary ligament

4 The left triangular ligament is very dense and suspends the left lobe of the liver to the diaphragm The left triangular ligament reinforces the falciform ligament

5 The round ligament is the fibrous remnant of the umbilical vein pedicle and is the antero-inferior suspension cable between anterior abdominal wall and the porta hepatis

6 The hepatoduodenal ligament is partly derived from reflections of the peritoneum from the lateral edge of the hepatic pedicle It is also composed of extensions of fasciae and connective tissue originating around the common bile duct duodenum pancreas, hepatic artery and portal vein

7 The hepatogastric ligament is a wide extension of the lesser omentum It includes the medial (left) edge of the hepatic pedicle It is also continuous with peritoneal and fascial reflections from the duodenum common bile duct portal vein and pancreas

These ligaments form a multistranded spider web which contains the liver mass One or several of these ligaments may be severed without abnormal incident Were they all to be severed the perivascular tissues of the portal and hepatic veins could support the liver until the fascial and peritoneal layers were reformed

The subdiaphragmatic space on the right consists of suprahepatic and infrahepatic divisions The suprahepatic (or subphrenic) space is divided into right and left sides by the falciform ligament The bare area between the dome of the liver and the diaphragm is a non peritonealized triangle between the falciform left and right triangular ligaments If intrahepatic abscess perforates the area of the bare space such suprahepatic collection is extraperitoneal These are potential spaces which become important when exudate or tumor is present

Infrahepatic collections may involve anterior or posterior right or left subhepatic spaces Anterior collections occur in the peritoneal cavity ventral to the stomach Posterior collections involve the lesser omental sac The foramen of Winslow (epiploic foramen) posterior to the hepatoduodenal ligament divides the posterior space into right and left sections The pyloroduodenal area divides the anterior space

2 Intrahepatic Ducts

Liver lobes have a fairly standard architecture with generally uniform distribution of the bile ducts The right lobe is drained by two intrahepatic bile ducts the left lobe is drained by one main duct

From a geographical maze representing the hepatic cells and its triple circulation there arises from a seeming fourth dimension a group of bile

canaliculi which emerge from lobular arborization to form interlobular cholangioles These drain the liver's hemispheres to form the left hepatic and two right hepatic ducts The left hepatic duct is centrally located At the liver hilum it disappears deep within the liver mass The right posterior hepatic duct is also deep and central within the liver mass The right anterior hepatic duct however, is more readily accessible From the hilum its course may be readily dissected because the major vascular arcades are posterior or inferior to it In addition, when it is dilated, because of a congenital atresia or by reason of organic obstruction in the common bile duct, it may be readily located by tangential incision into (the hepar lobatum when it is present or) the anterior inferior aspect of the right lobe The lumen of the left hepatic duct is generally of wider diameter than the right even though the left lobe represents the smaller mass

The left main duct and the two right branches of the hepatic duct join deep within the porta hepatis at the level of the bifurcation of the portal vein Usually the common hepatic duct swerves anteriorly and to the right over the right branch of the portal vein In some cases the common hepatic duct may angle upon itself up to 60 degrees as it crosses the portal vein in the lateral aspect of the hepatic pedicle

Interrelationship among the bile capillaries and the three hepatic vascular trees is amazing Three tubular systems enter the liver hilum inferiorly 1) The hepatic artery accompanies the portal vein in its anatomical distribution The arterial supply tends to be segmental with few or no interlobular anastomoses 2) The portal vein centrally located amidst the functioning hepatic cells develops many sinusoids towards its terminations Its flow is almost invariably segmental splenic, gastric and pancreatic blood is diverted to the left lobe whereas intestinal blood enters the right lobe 3) The third hepatic circulation is venous It is in intimate contact with the bile canaliculi The larger hepatic veins and the bile capillaries are in close juxtaposition

Occlusion to the hepatic artery can be tolerated by the liver for 15 minutes Occlusion to the portal vein can be permanent provided that the venous flow is diverted Occlusion to the hepatic veins can be tolerated for one hour without fatality The bile duct's occlusion may persist for months prior to fatality —for years if some constant decompression is provided

The volume of the liver is approximately 1500 grams The contained blood occupies approximately 800 cc (approximately 20 per cent of the total blood volume) Of the blood contained in the liver approximately 100 cc is arterial about 200 cc is hepatic venous and approximately 500 cc is portal in origin Bile present in the capillaries, cholangioles and hepatic ducts does not normally exceed 20 cc

Hepatic cells are arranged as plates which form the "lacunae " The

hepatic lobules are the areas which surround the central veins and are continuous with one another (Elias)

The liver parenchyma is tunneled by "portal canals" which are surrounded by a sheet of cells (periportal limiting plates) Each portal canal contains a branch of the portal vein, a branch of the hepatic artery a network of bile ducts a network of lymph vessels and perivascular nerves

From the portal vein inlet venules penetrate holes in the canals and ramify into sinusoids which empty into central veins The central veins unite with sublobular and large hepatic veins

From the hepatic artery, arterioles and capillaries enter the lobules at different levels One set empties into periportal sinusoids and another set empties into the sinusoids near the center of the lobules Prolonged contraction of arterioles may cause zonal anoxia (Zahn)

The central and sublobular veins are at right or oblique angles to the portal canals Where the limiting plate is pierced by a venule, the perisinusoidal spaces (Disse) are continuous with the periportal tissue space (Mall) From here tissue fluid seeps through the periportal connective tissue into the lymphatics

The bile capillaries form a polygonal network within the liver plates between hepatic cells The bile canaliculi have a cellular wall Minute bile ducts enter the lobules and receive bile from intralobular plates and then form loops or networks Most of the bile drains through the canalicular network through limiting plates and the canals of Hering into the bile ducts

Compression phenomena are interdependent In passive congestion from heart failure increase in hepatic vein volume may compress bile capillaries *Marked dilatation of the bile capillaries can decrease the hepatic* blood volume This is of such importance that care must be exercised in decompressing the bile duct at surgery (and postoperatively) for relief of prolonged obstructive jaundice Two hundred to 400 cc of blood may be lost from the circulating blood volume into the liver following sudden decompression of bile passages

B Extraduodenal Biliary Ducts (Figs 3 and 4)

1 Hepatic Duct

The junction of left and right hepatic ducts is usually at the midpoint of the hilar margin for the quadrate lobe It tends to be toward the right if it is not located in the midline The hepatic ducts form a wide angle of convergence Major tributaries may *empty into the superior aspect of the* diverging angle and parallel each other

Right and left hepatic ducts vary in length from 0 7 to 3 0 cm These merge just after leaving the liver hilum to form the common hepatic duct



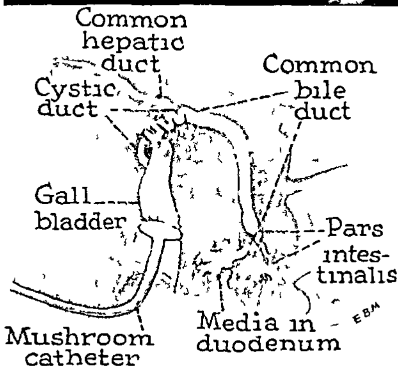


FIG 3 CHOLECYSTOCHOLANGIOGRAM

Common hepatic duct not filled well Gallbladder fills without abnormality Cystic duct common bile duct and termination to duct are normal Unimpeded flow of contrast media into duodenum

In only 12 to 15 per cent of cases do both hepatic ducts continue individually beyond 2 cm from the liver hilum In about 5 per cent of cases the common hepatic duct alone emerges from the hilum, the branches have joined within the liver

As the ducts emerge from the hilum to form the common hepatic duct they are covered by a strong fibrous sheath This merges with areolar tissue of the hepatic pedicle (hepatogastric and hepatoduodenal ligaments) and separates the ducts from adjacent blood vessels The common hepatic duct lies to the right and is anterior to branches of the (right) hepatic artery

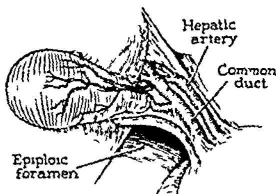


Fig 4 Hepatic Pedicle

Between the liver and the pyloro duodenum are reflections of the omenta the hepatic pedicle It contains the hepatic artery and its branches the common bile duct the common hepatic duct the cystic duct termination the portal vein lymphatics nerves and areolar tissue The right border of the hepato duodenal ligament is the anterior superior boundary to the foramen of Winslow

Posterior to the hepatic artery as well as to the common hepatic duct lies the portal vein which divides into right and left branches together with the ducts The left branch of the portal vein and the left hepatic duct enter the left lobe of the liver in the umbilical fissure at a point between the caudate and quadrate lobes

The common hepatic duct length depends upon the level of union with the cystic duct Its usual length of 3 0 to 3 5 cm does not indicate that the hepatic duct may normally be absent when the cystic duct joins the right hepatic duct or the hepatic duct be abnormally long if the cystic duct parallels the hepatic duct for 80 per cent of its extraduodenal course Both variations occur The common hepatic duct is a flexible tube about 0 5 cm in diameter The right hepatic duct is found as the accessory duct in approximately 18 per cent (fig 22) Accessory ducts may enter the gall bladder the cystic duct or the common bile duct

investment of the gallbladder covers 60 per cent of the surface area The other 40 per cent of the gallbladder wall rests immediately adjacent to hepatic tissue in a spoon shaped fossa which has a depth of approximately 2 5 cm Variations occur in size of the gallbladder fossa The fossa may be absent when the gallbladder peritoneum acts as a mesentery The fossa may be huge if the gallbladder is intrahepatic

The size of the normal gallbladder varies with its contents It may extend to the liver margin or may be above or below it Fifty five per cent are inframarginal, 30 per cent may be marginal and up to 15 per cent can be supramarginal The normal gallbladder in resting state usually contains 30 cc The gallbladder may normally expand to contain over 100 cc This volume is decreased by fluid (water) absorption (chapter 3)

The normal gallbladder may have an "ampulla" When present this section of the gallbladder is adjacent to the cystic duct area It absorbs the effect of the fluid wave due to gallbladder contraction Because the posterior and medial aspect of the gallbladder is adjacent to the liver the ampullary dilatation is present on the free external surface Structurally another ampulla, actually the so called Phrygian cap is identified at the fundus It is most prominent in supramarginal gallbladders

4 Extraduodenal Common Bile Duct

There are two major segments to the extraduodenal common bile duct The proximal portion originates at the union of the common hepatic and cystic ducts, anterolateral to the portal vein, within the hepatic pedicle The distal portion leaving the hepatic pedicle passes posterior to the duodenum and frequently through the pancreas to enter the duodenum It travels in a three dimensional long, flat "S" shaped curve (fig 6) Together, the common bile and hepatic ducts range from 9 to 20 cm in length Usually the common hepatic duct is 3 cm and the common bile duct 10 cm long

The hepatic pedicle segment of the common bile duct is located in the median sagittal plane and in the middle third of the right half of the body between the 12th thoracic and second lumbar vertebrae It passes through the hepatic pedicle in a slight posteromedial curve The largest diameter of the common bile duct is 6.3 mm (table 1) The average thickness of the wall (table 2) is 0 7 mm

There is an abrupt change to antero-inferior direction by the common bile duct from the hepatic pedicle toward the pancreas and duodenum When the common duct passes through the pancreas its walls are rigid and narrow In 33 per cent the distal portion of the extraduodenal common bile duct is extrapancreatic throughout In 31 per cent the duct is totally intrapancreatic In the remaining 36 per cent the common bile duct is



FIG 6 CHOLANGIOGRAM

Normal contour and position of the bile ducts is seen The transpancreatic segment of the extraduodenal bile duct has a rigid (and decreasing) lumen The transduodenal segment is well visualized as a filamentous curving stem to a funnel (Courtesy of Arch Surg)

partially intrapancreatic being covered by a thin shell of pancreas (figs 7a b and c)

The intima of the common bile duct is smooth and grayish yellow It is marked by flecks which are a fraction of a millimeter in diameter These are the depressed orifices of one to four intramural glands per square centimeter of duct surface Under the microscope the epithelial lining is seen as a layer of low columnar epithelial cells with keratin like deposits and bile pigment on the surface There is a submucosal space crossed by glandular orifices and a fibrous connective tissue layer containing very little smooth muscle In 7 per cent of cases there is no smooth muscle found in the bile ducts The outer layer of epithelium contains many lymphatics and vessels

Histological structures of the common cystic (distal part) and hepatic ducts are identical The bile duct lining is covered with a layer of tall columnar epithelium The nucleus at the base of the cell is large vesicular and well stained Epithelium is similar to that which covers the rugae of the gallbladder The epithelial layer frequently desquamates within an hour after death due to enzyme activity The connective tissue layer of the duct just beneath the lining epithelium contains considerable elastic tissue The connective tissue layer is thick and compact The outer coat of the duct

TABLE 1 DIAMETER OF EXTRADUODENAL COMMON BILE DUCT

Number of Cases	Diameter
	mm
3	3 2
5	4 0
9	4 8
1	5 2
1	5 6
13	6 4
4	7 2
5	9 6
1	10 4
2	11 9
1	12 7
Total 45	6 3 (average)

TABLE 2 COMMON BILE DUCT WALL THICKNESS

Number of Cases	Thickness
	mm
16	0 4
22	0 8
2	1 2
2	1 6
Total 42	0 7 (average)

is composed of a loose layer of areolar connective tissue in which are found blood vessels lymphatics muscle and nerves The muscularis in the outer layer of the wall is composed of unstriped fibers in isolated longitudinal and circular bundles The longitudinal fibers are larger and better developed A cross section will usually reveal three or four large round bundles equidistant *in the periphery of the duct, separated by connective* tissue which may contain several smaller muscle bundles Muscular tissues are very sparse in the ducts except at the pars intestinalis

C Transduodenal Bile Duct (*Pars Intestinalis*)

Certain studies of the common bile duct indicate that anatomical variation exists in blood vessels lymphatics, nerves and surrounding tissue However, more complete investigation has shown that the anatomical course and characteristics of the common bile duct are standard and uniformly recognizable with few variations

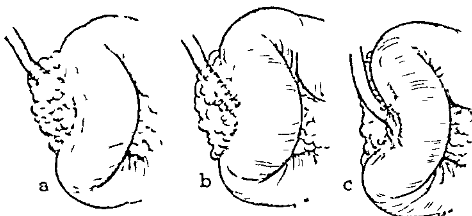


FIG 7 RELATIONSHIP OF COMMON BILE DUCT TO PANCREAS

A The distance between the duodenum and the common bile duct proximal to the *pars intestinalis* is maximal when the termination of the extraduodenal common bile duct is totally transpancreatic

B The interval between duodenum and bile duct is less when the bile duct is partially pancreatic and/or covered by extensions from the capsule of the pancreas

C The space between the bile duct and the duodenum is least when the duct is extrapancreatic and adjacent to the duodenal wall

This also applies to the termination of the common bile duct It is composed of several groups of sphincter muscles The sphincter is part of the hepatopancreatic duct in the embryo It originally appears as a primitive ampulla which envelopes several openings into the duodenum (Boyden) It is co-extensive with hepatopancreatic buds and the duodenum An obliquity of bile structures passing through the duodenal wall is recognizeable (fig 13) The bile ducts originate from channels formed by the coalescence of vacuoles following the nearly solid stage in the development of the duodenum (Arey)

Beginning at the 20 mm stage the junction of bile and pancreatic ducts recedes from its window in the intestinal wall into the submucosa At maturity of the embryo this ampulla has involuted and a center of interstitial growth has expanded in the pre ampullary segment of the ducts which forms the sphincter of Oddi The embryonic ampulla does not (usually) exist beyond the duodenal submucosa

The first sign of a musculus proprius is a concentric arrangement of the mesenchyma about the bile and pancreatic ducts in a 26 mm embryo This encircles the pre ampullary portion of the ducts just beyond the point where they have pierced the tunica muscularis

Between the 41 and 45 mm stages, four weeks after the intestinal muscle has formed this concentric mesenchyma differentiates into the pre ampullary zone of intrinsic muscle It appears about the same time as the

first rings of smooth muscle that encircle the ureter Similarly, it has a characteristic growth gradient whereby the muscle fibers differentiate successively in the direction of the papilla

Superimposed upon this is a secondary center of interstitial growth located in the pre ampullary segment of the ducts As growth moves caudal it leaves behind it a sphincter (superior) located in the intestinal window and a sphincter (inferior) along the intramural portion of the bile duct It also pushes the junction of the bile and pancreatic ducts further into the submucosa thereby reducing the relative length of the ampulla This movement is aided by the rapid growth of the intestinal mucosa the buckling of which produces the plica longitudinalis This sequence of growth derives its chief significance from the fact that it carries the main sphincter of the bile duct away from the intestinal muscle and sets it up as an independent mechanism for regulating the flow of bile

The major papilla varies in its location on the postero or posteromedial duodenal wall *from 55 to 142 mm (mostly from 80 to 120 mm) from the* pylorus *In 54 per cent of cases the papilla is elevated above the* surrounding *mucous membrane In 22 per cent of cases it is level* with the surrounding duodenal mucosa In 24 per cent of cases the orifice of the common bile duct is visible in a depression surrounded by irregular ridges of duodenal mucous membranes

It is obvious, then that in 76 per cent the papilla may be visible as a prominence However although in the remaining 24 per cent it is depressed it is still composed of a solid mass of tissue

Although the duct of Wirsung is medial and inferior to the common bile duct that relationship is not always maintained because the ducts may intertwine The termination of the pancreatic duct in 42 per cent of cases is almost directly inferior and slightly medial to the orifice for the common bile duct The other 58 per cent of cases show a scattering in location of the exit for the pancreatic duct If papillotomy or sphincterotomy be done it is essential to identify the interrelations of the pancreatic and common bile ducts orifices

The external appearance of the common bile duct is deceptive To superficial examination of the duct termination the diameter increases distal to the apparent junction of the pancreatic and common bile ducts This enlargement gradually decreases to a blunt ending within the duodenal canal (fig 8)

However sagittal section through the duct reveals that the increase in size seen externally is not due to an increase in lumen diameter Actually it is due to the increased width of the common duct wall There is a decrease in the diameter of the lumen as the common bile duct crosses the duodenal wall (fig 9)

The lumen of the pancreatic duct changes similarly but with not as much contrast because there is a smaller diameter and proportionately less (sphincter) muscle tissue

The thickness of the duodenal wall varies from 3 to 12 mm Its average thickness is 5 mm By contrast the papilla is from 7 to 10 mm in length

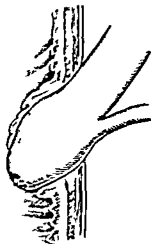


FIG 8 EXTERNAL APPEARANCE OF THE PAPILLA

The entrance of the pancreatic duct is proximal to a rounded swelling at the termination for the bile duct Orifices for the common bile duct and pancreatic duct are *identified at the apex of the papilla within the duodenum*

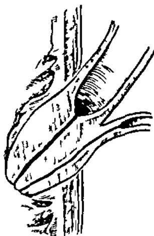


FIG 9 LONGITUDINAL SECTION THROUGH THE PAPILLA

The increased size of the common duct at the papilla is due to thick (sphincter) tissue In the papilla the lumens for common bile and pancreatic ducts decrease in diameter An ampulla (or a dilatation in the duct lumen) is uncommon (? per cent) although normal

TABLE 3. SIZE OF THE PAPILLA (LENGTH AND RADIUS)

Number	Length
	mm
1	7.2
1	8.8
1	9.6
1	10.4
2	11.0
1	11.9
9	12.7
1	13.5
7	14.3
11	15.9
2	16.5
1	17.0
1	17.4
1	18.2
4	18.9
Total 44	14.1 (average)
Number	**Radius**
	mm
1	1.6
4	2.2
7	2.6
15	3.4
3	4.2
2	5.0
3	6.4
1	9.6
Total 36	3.4 (average)

averaging 1.4 cm in length. At its widest axis the diameter of the papilla ranges from 3.2 to 19.2 mm, averaging 6.8 mm (table 3).

The transduodenal course of the common bile duct is transpapillary. In general the duct, through this region, traverses a flattened "S" shaped route which curves on more than one axis (fig. 10). The common bile duct enters the duodenal wall at somewhat less than a 90-degree angle. As it passes through the muscular layer of the duodenal wall there is a slow curve which parallels the longitudinal axis of the duodenal canal. At the termination the axis of the duct turns again to empty into the duodenum at nearly a right angle (fig. 13).

The greatest decrease in diameter occurs in the proximal third of the

transduodenal common bile duct This can best be evaluated by cross section study

Proximally the two ducts are completely separated Circumferential muscle and fibrous tissue totally surround the common bile duct the pan



FIG 10 POSTOPERATIVE CHOLANGIOGRAM

The transpapillary segment (*pars intestinalis*) of the extra duodenal common bile duct is visualized on this radiograph Its average length is 14 mm It may be normal if the pars intestinalis is not visualized (This cholangiogram was done after six months of Fribram therapy There are no residual calculi Patient is well after seven years) (Courtesy of *Ann Surg*)

creatic duct is loosely attached to the pericholedochal tissues by fine areolar and fibrous fatty tissue (fig 11)

In the middle of the papilla a rim of muscular and fibrous tissue surrounds each duct and also surrounds both of these sphincter muscles The duct diameter diminishes as the extent of this muscle increases Terminally the ducts come together the interductal muscle is gradually lost At their tips only loose epithelial tissue is present

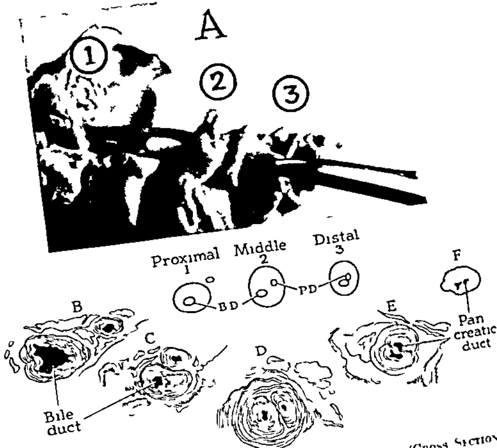


FIG. 11. TERMINATION FOR THE BILE AND PANCREATIC DUCTS (CROSS SECTIONS)

CD, common bile duct; PD, pancreatic duct. (Courtesy of *Am. J. Gastroenterol.*)

A. Sketch indicates characteristics of different levels through the termination for the common bile and pancreatic ducts. 1) Proximal: the pancreatic duct is outside the thickened common bile duct. 2) Middle: the pancreatic duct and common bile duct are enclosed within the same tissues. 3) Distal: the common bile duct lumen is smaller, the two ducts are contiguous, and the thickness of the surrounding tissue has decreased.

B. Extraduodenal: the pancreatic and common bile ducts are seen outside the duodenal wall approximately 15 mm from the orifice of common bile duct. Each duct is separate. The pancreatic duct has a wider lumen, a thicker wall, and is partly surrounded by pancreatic tissue.

C. Proximal papilla, approximately 10 mm from the orifice of the duct. The ducts are in the outer wall of the duodenum. Individual groups of circular muscle surround each duct. The pancreatic duct is small, thin, and has a smooth mucosa; the common bile duct is larger, has a thicker wall and a more irregular mucosa. Occasional fibers from the duodenal musculature merge with muscle surrounding the duct. Each duct maintains identities.

D. Middle papilla. Bile and pancreatic ducts

Close observation of the duct lining reveals a sharp difference between the extraduodenal and transduodenal segments of the common bile duct.

The translucent intimal membrane of the extraduodenal common bile duct is pock marked with about three to four orifices per square centimeter. However, in the papillary portion, longitudinal or oblique striae and pleats are found. In most specimens (95 per cent) folds and reduplications are seen which look like valvules (fig 12). These are 2 to 4 mm long, 1 to 2 mm high and less than 1 mm in thickness. They are irregular in axis and direction. The edges of one fold may merge with the midportion or edge of another. Many small folds can appear within a larger cusp. The arc of the valvule may point in either hepatic or duodenal direction. On microscopy, muscular and fibrous tissues are found extending from the wall of the duct in the valvule (Sterling).

On x ray, the average length of the transduodenal segment of the duct is 14 mm. The lumen of the common bile duct decreases in diameter as it crosses the duodenal wall. Its sides have a double concavity or scalloping which gives the lumen the appearance of a funnel. The duct finally terminates as a thin filamentous canal, the orifice of which measures approximately 2 mm in diameter (fig 13).

D The Common Channel for Bile and Pancreatic Ducts

There has been much controversy concerning the incidence, character, significance and function of the common channel for the common bile and pancreatic ducts terminations. Over 1200 specimens reported by 13 investigators varied between 11 and 80 per cent in the incidence of separate orifices for the common bile and pancreatic ducts (table 4).

To clarify this problem, special studies were completed without damaging the papilla. Studies were done on transparent specimens after injection of radio-opaque and colored dye. This study has been very illuminating.

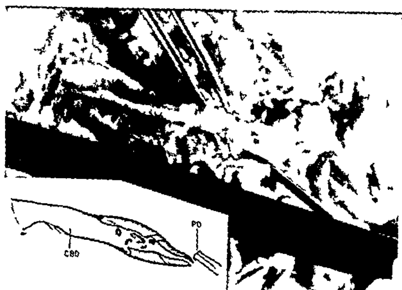
Separate orifices for both common and pancreatic ducts are observed in 37 7 per cent. In 3 per cent only the common bile duct traverses the papilla of Vater. In these cases the pancreatic duct is atrophied and the pancreas empties only through the accessory pancreatic duct. Thus in 41 per cent there is no possible intermingling of pancreatic and biliary tracts (fig 14).

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surrounded by circular muscle. Peripherally, muscle bundles are enclosing the individual duct circular muscles.

E Distal papilla approximately 3 mm from the termination. The ducts are separated by a thin fibro muscular septum.

F Termination of papilla approximately 0 5 mm from the termination of the ducts in the duodenum. Duodenal mucosa is penetrated by orifices for the common bile and pancreatic ducts. At this level the septum between the two ducts is composed of the ducts' walls.



A



B

FIG 12 VALVULES IN TERMINATION OF BILE DUCT

A Photograph of common bile duct after longitudinal incision through the papilla A ruler parallels the bile duct A probe passes into pancreatic duct through a channel common to both ducts for 2 mm The wall of the extraduodenal bile duct is thinner and its lumen much wider than the transduodenal (*pars intestinalis*) segment The mucosa of the extra duodenal duct is smooth except for stippling produced by glandular orifices The transduodenal bile duct mucosa is reduplicated and valvules are seen Insert is diagram of the photograph

B Histologic section through a valvule reveals fibrous and muscular tissue together with glandular and epithelial structures



FIG 13 PARS INTESTINALIS

Upper Radiograph of cadaver study after injection of diodrast® into common bile duct

Lower Subsequent radiograph taken after additional injection of lipiodol into pancreatic duct Multiple pancreatic ducts orifices may be observed in this case

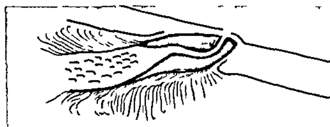


FIG 13—*Continued*

Diagram of *pars intestinalis* to indicate its curving funnel like lumen with at least three changes in direction

TABLE 4 DISSECTION REPORTS OF ORIFICES FOR COMMON BILE AND PANCREATIC DUCTS

Investigator	Number of Specimens	Separate Orifices	Percentage
Baldwin (1911)	90	20	22
Belou (1915)	50	27	54
Cameron, Noble (1924)	100	26	26
Dardinski (1936)	100	51	51
Howard (1947)	150	41	27
Hozapfel (1930)	50	40	80
Letulle Nattan (1898)	21	15	71
Mann Giordano (1933)	200	62	31
Opie (1903)	100	11	11
Reinhoff Pickrell (1945)	250	73	24
Ruge (1908)	43	11	26
Schirmer (1893)	48	22	47
Sterling (1948)	50	32	64
Totals	1252	359	29

A common channel is present between the terminations of the common bile and pancreatic ducts in 59 per cent. In 44.3 per cent a common channel *is in the distal third of the papilla (fig 15)*. *In 13.1 per cent the common* channel for the common bile and pancreatic ducts traverses half of the papilla of Vater (fig 16). *In only 1.6 per cent does the common channel* cross nearly the entire papilla of Vater (fig 17). It is possible for reflux or *regurgitation between pancreatic enzymes and bile to occur only in 15* per cent from the anatomical point of view.

The largest group (44 per cent) is composed of those in which a small common channel is formed between the common bile and pancreatic ducts distal to the sphincter muscles of the papilla of Vater. The average length of the common channel is 0.3 cm. It is difficult to distinguish this common channel from overlying duodenal mucous membrane. Under some circumstances any of this group of specimens might be considered as having either a single orifice or two openings depending upon the character of the interductal septum.

In 60 per cent the common bile and pancreatic ducts merge to form a common channel from 0.1 to 0.8 cm in length. However, in these specimens the average length of the common channel is 0.4 cm as compared to the average length of the papilla of 1.1 cm (table 5).

It is found that in 75 per cent the common channel traverses only the distal third of the papilla. In 22 per cent the common channel is in the distal two-thirds of the papilla. In only 3 per cent does the common channel occupy more than two-thirds of the papilla.

There are minimal variations in the diameters of the orifices for common bile duct, pancreatic duct and the common channel. The diameters of the orifices range from 0.05 to 0.2 cm. The smallest average diameter in the pancreatic duct is 0.01 cm. The common bile duct average orifice is 0.13

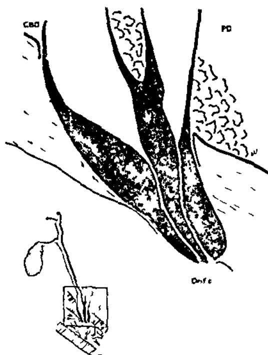


Fig. 14. Separate Orifices (41 Per Cent)

The papilla has two separate orifices. Sphincter tissue is uniformly distributed around both pancreatic and common bile duct, decreasing at the termination of the ducts.

Figures 14, 15, 16 and 17 are line drawings of transparent injected specimens of the papilla of Vater. The common bile and pancreatic ducts are illustrated in their courses through the papilla and in relation to the duodenal wall in the four normal types of ducts terminations. CBD, common bile duct; PD, pancreatic duct; CC, common channel. Lined area, duodenal wall; curved line area, pancreas; cross hatched area, papilla (sphincter muscles). (Courtesy of *Surg., Gynec. & Obst.*)

cm. The average diameter of the orifice for the common channel is 0.17 cm. The diameter of the orifice for the common channel is greater than the diameter of the orifices for either the common bile or pancreatic ducts.

It is found to be just as common for a slight dilatation to occur in the course of the common bile or pancreatic ducts as in the route of the common channel. An area of dilatation may be present at a turn in the bile or pancreatic duct axis or within the papilla where sphincter muscle distribu-

tion is altered These dilated areas are slight as compared to the length and breadth of the papilla and its contained ducts An actual "ampulla" within the papilla of Vater is rare and unusual but not abnormal, per se

The duodenal wall hiatus for the entrance of the common bile duct is at the posteromedial aspect adjacent to the head of the pancreas and in a segment of the duodenal wall which is between the two vascular arcades This space is not covered by peritoneum although areolar tissue and pan

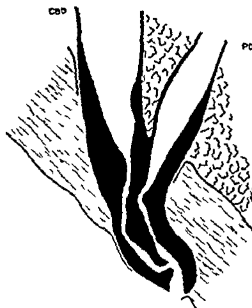


Fig 15 Common Channel in Distal Third of Papilla (11 Per Cent)

There is a single orifice The papillary muscle tissue is well developed The ducts diameters rapidly decrease The ducts are parallel through at least two thirds of their trans papillary courses The axis of each duct is a flat S shaped curve At about the junction of middle and distal thirds of the papilla the common bile duct turns at a right angle to enter with the pancreatic duct, into a shallow common channel The common channel may vary from 0.1 to 0.6 cm in length

creatic capsule are immediately contiguous This hiatus in the duodenal wall is associated with a split in the longitudinal and circular duodenal muscular coats Occasionally fibers from these muscular coats are adherent to the papilla

The papilla and the adjacent sleeve of tissue has a range of movement which normally is 1 or 2 cm It is not abnormal for movement of the papilla to occur to the opposite duodenal wall Normal mobility under probe guidance can occur for greater than 3 cm

The papilla occasionally can be palpated The presence of a probe or catheter in the extraduodenal common bile duct propels the papilla forward

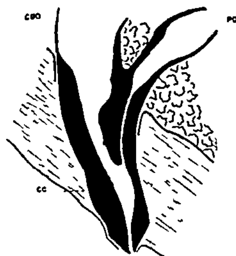


FIG 16 COMMON CHANNEL THROUGH DISTAL HALF OF THE PAPILLA (13 PER CENT)

There is a single orifice at its apex At the entrance of pancreatic and common bile ducts into the papilla their lumens decrease Toward the middle of the papilla the ducts merge and there is a widening in the diameter of the common channel

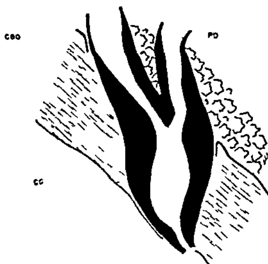


FIG 17 COMMON CHANNEL THROUGH MOST OF THE PAPILLA (2 PER CENT)

The common channel for pancreatic and common bile ducts traverses more than half of the papilla The papilla is conical and firm with a single orifice There is well developed muscle in the proximal half of the papilla There is a gradual decrease in the diameter of the common bile duct as it crosses the proximal portion of the papilla The pancreatic duct decreases in diameter The pancreatic duct joins the common bile duct at the junction of the proximal and middle thirds of the papilla to form a common channel The dilatation present at the common channel is an ampulla

TABLE 5 RELATION OF COMMON CHANNEL TO PAPILLA

Length of Common Channel	Length of Papilla	Papilla Was Larger than Common Channel by
mm	*mm*	*mm*
1 2	3 2	2 0
1 6	14 3	12 7
2 0	11 0	9 0
2 4	13 5	11 1
2 5	16 9	14 4
3 2	15 9	12 7
3 6	12 7	9 1
4 0	14 3	10 3
4 0	15 9	11 5
4 0	18 2	14 2
4 8	15 9	11 1
4 8	destroyed by dissection	
6 4	15 9	9 5
6 4	15 9	9 5
6 4	15 9	9 5
6 4	18 9	12 5
6 4	18 9	12 5
8 4	8 4	0
Average 4 4	14 4	10 1

and permits some accuracy in localization of the papilla However, localization of the papilla by this method should be considered to be at least 2 cm too short

In summary (table 6) the following facts are available regarding the anatomy of the choledochus and its papilla of Vater

1 The common bile duct pursues a constantly curving course
2 The termination of the common bile duct in its transduodenal segment as the papilla of Vater
   a is like a funnel in that its lumen rapidly decreases in size (and rate of flow proportionately increases)
   b curves through a three dimensional route frequently intertwining with the pancreatic duct
   c unites with the pancreatic duct to form an anatomic "common channel" in 50 to 60 per cent of cases
3 A common channel which traverses less than one half to two thirds of the papilla of Vater does not really permit interductal reflux because of the anatomic disposition of the sphincter masses Thus a physiologic common channel exists only in approximately 5 per cent of cases

TABLE 6 SIGNIFICANT MEASUREMENTS OF THE BILE DUCTS

	cm
Diameter of extraduodenal common bile duct	
Median	0.5
Average	0.56
Length of papilla	
Median	1.5
Average	1.47
Widest thickness of wall of papilla	
Median	0.6
Average	0.69
Length of transpapillary common bile duct	
Median	1.2
Average	1.26
Length of transpapillary pancreatic duct	
Median	1.0
Average	0.89
Diameter—orifice of the common bile duct	
Median	0.1
Average	0.12
Diameter—orifice of the pancreatic duct	
Median	0.1
Average	0.09
Diameter—orifice of the common channel for the common bile and pancreatic ducts	
Median	0.1
Average	0.1

F The Duodenum

The duodenum has four anatomical divisions between the pylorus and the duodenojejunal flexure It is 25 to 30 cm long and in general has a C shape with an elongated lower limb The pancreas is wedged within the convexity of the 'C

Except for its terminal several centimeters the duodenum lies at the upper right supramesocolic section of the peritoneal cavity Except for the first portion the anterior wall of the descending limb and the terminal several centimeters the duodenum is retroperitoneal

The cap or first portion of the duodenum is intraperitoneal It passes right posterocraniad from the pylorus between the approximate level of the first or second lumbar vertebra It is mobile

The duodenum curves convexly between the cap and the descending limb This second division of the duodenum is posterior to the plane of the hepatoduodenal ligament and anterior to the hilum of the right kidney Posteriorly the duodenum at this area is extraperitoneal Extensions from the hepatic flexure and ascending colon convert the terminus of the de

scending duodenum and its consequent course as third or transverse portion to an extraperitoneal location

The medial and posterior aspects of the descending duodenum caress the pancreas and the terminal portion of the extraduodenal common bile duct Penetrating the double vascular arcade *along the posterior medial aspect* of the duodenum through a hiatus in the duodenal wall are both common bile and pancreatic ducts which join through the papilla of Vater to empty within the duodenum An accessory pancreatic duct enters similarly some several centimeters superiorly to the papilla of Vater Smaller pancreatic ducts also empty in the descending duodenum This limb is fixed in position by its peritoneal reflection The peritoneal reflection is fused together with that of the head of the pancreas to the posterior parietal peritoneum Variations in its fusion to the posterior abdominal wall modify the mobility The duodenum is also suspended by means of the hepatic pedicle (hepatogastric and hepatoduodenal ligaments) and its contained bile duct and portal vein Perivascular fibroareolar tissues accompanying the pancreaticoduodenal arcades also assist in fixation of the descending duodenum

The third or transverse division of the duodenum runs horizontally and slightly upward across the ureter, inferior vena cava and vertebral *column* It turns anteriorly and caudad at the level of the second or third left lumbar transverse process The transverse duodenum is posterior to (*from right* to left) the inferior aspect of pancreatic head the inferior pancreaticoduodenal artery the superior mesenteric vein and artery and the root of the mesentery to the jejunum

The fourth division of the duodenum is to the left of the vertebral column It curves around the left side of the root of the transverse colon mesentery There are superior and inferior folds of peritoneal reflections at the junction *of duodenum and jejunum* The peritoneal reflections (right left and superior) to the diaphragmatic crus and the posterior abdominal wall compose the ligament of Treitz

The blood supply to the duodenum is provided by the superior and *inferior pancreaticoduodenal arteries and the gastroduodenal artery* (Michels) These are in the form of arcades, ventral and dorsal to the head of the pancreas There is only one collateral marginal arcade Anastomosis adjacent to the duodenal wall and an inconsistent intramural plexus is *present Veins which accompany the arteries drain into the* superior mesenteric, lienal and anterior and posterior pancreaticoduodenal Innervation is profuse from the celiac ganglia

F The Pancreas

The pancreas arises from the primitive gut below the stomach at the level of the hepatic primordial bud (fig 18) A ventral element arises from



FIG 18 DIAGRAMS OF THE HUMAN PANCREATO BILIARY SYSTEM IN THE EMBRYO (AFTER ARLY)

A 8 mm embryo relationships are represented between the duodenum the dorsal and ventral pancreas to the biliary hepatic duct

B In the 12 to 16 mm stage dorsal and ventral pancreatic buds coalesce Intra pancreatic duct arborizations merge

the hepatic bud The dorsal pouch arises from the duodenum The ventral and dorsal segments merge Cephalic portions of the head the neck body and tail of the pancreas arise from the dorsal anlage The caudal portion of the head and uncinate process migrate dorsally to fuse with the dorsal segment

The main pancreatic duct arises from the ventral diverticulum as a secondary outpouching of the hepatic primordium It is contiguous to the common bile duct The duct of the dorsal embryonic element becomes the accessory pancreatic duct (Santorini) and empties directly into the duodenum This duct may remain patent as a tributary of the main pancreatic duct (fig 18b)

The pancreas in early embryonic development consists of arborizing tubules As growth by progressive duct division continues some of the cellular elements differentiate to form acinar and islet tissue These specialized cellular structures always develop in relationship to ductal elements although the ducts within the islet become impervious

The pancreas is a soft elongated yellowish pink lobulated glandular structure extending from the concavity of the duodenum to the hilum of the spleen The pancreas consists of a head neck body and tail The head and neck merge into an uncinate process The body of the pancreas is a triangular prism which tapers to a flat oval as it crosses vertebrae towards the superior aspect of the left kidney and then thins out to approach the hilum of the spleen The head and uncinate process is molded to the convexity of the duodenum and pylorus The inferior border of the gland to the right is notched by the superior mesenteric and portal veins The posterior surface of the head is also in apposition with the inferior vena cava the right ovarian (or spermatic vein) the right renal vessels and the aorta

The anterior surface of the pancreas is covered by the transverse mesocolon and/or the first portion of the transverse colon It is also covered by posterior parietal peritoneal reflections and multiple septa to the posterior wall of the stomach

The posterior surface of the pancreas crosses the aorta over the superior mesenteric artery and the left renal pedicle The splenic vein is posterosuperior to the pancreas The splenic artery is usually on the superior border of the distal part of the pancreas These vessels may be duplicated

There are one principal and one large accessory pancreatic ducts The main duct (Wirsung) extends throughout the central portion of the tail and body through the neck, and passes obliquely downward Pancreatic tissue is closely adherent as it joins with the common bile duct to enter the papilla of Vater and to terminate within the duodenum The other duct (Santorini) is usually a lateral branch of the duct of Wirsung It may be separated and drain the head and superior portion of the pancreas The point of emergence for this duct may be at anywhere from 1 to 4 cm proximal to the papilla of Vater There is usually found in conjunction with its entrance to the duodenum a thickening or papilla which like the papilla of Vater enters the duodenum through a small hiatus in oblique fashion In about 3 per cent of cases the duct of Wirsung is atrophied and the entire external secretion of the pancreas enters the duodenum through the minor papilla for the duct of Santorini

G Blood Supply

1 Biliary Tract

The blood supply of the biliary tract is generous and follows a pattern similar to the intestines The common hepatic artery proper (not the hepatic artery) is the major source The most frequent arteries supplying the tract are (a) posterior superior pancreaticoduodenal (100 per cent) (b) right hepatic artery (84 per cent) (c) posterior inferior pancreaticoduodenal (56 per cent) (d) right gastric (44 per cent), (e) hepatic artery (40 per cent) (f) anterior inferior pancreaticoduodenal artery (32 per cent), (g) cystic artery (28 per cent) (h) supraduodenal artery (24 per cent) and (i) superior pancreaticoduodenal artery (24 per cent) Basically these vessels are branches of the common hepatic trunk (common hepatic artery proper) As a rare anomaly this comon hepatic (proper) artery trunk is absent and the hepatic artery or the right hepatic artery will arise from the celiac axis or from the superior mesenteric artery The closest vessel supplies portions or all of the biliary tract

From the common hepatic trunk (proper) there arises the hepatic artery and the gastroduodenal artery From the hepatic artery there arises the right gastric artery, the left and right hepatic arteries From the gastro

duodenal artery are derived the anterior and posterior pancreaticoduodenal arteries The supraduodenal artery (Wilkie Michels) may arise from the gastroduodenal artery may be duplicated or may arise from other source

The hepatic artery in over 90 per cent branches into right and left hepatic arteries Additional hepatic arteries (right and left) are present normally in approximately 20 per cent These arise from the superior mesenteric artery the celiac axis and the cystic artery

The hepatic arteries are normally medial inferior and posterior to the bile ducts The terminal branches of the left hepatic artery are to the left of the ducts those of the right hepatic are to the right of the associated ducts The right and left hepatic arteries divide before entering the hilum Each hepatic artery may enter the liver as two branches (25 per cent) three branches (40 per cent) and four five or no immediate branches (30 per cent) Abnormal situations pertain in about 5 per cent

It is normal for the right and left hepatic arteries to send branches to the opposite liver lobe in from 10 to 30 percent of cases

The portal vein is a single trunk throughout the entire hepatoduodenal ligament It originates beneath the pancreas Liver branches do not ramify until within the hilum Occasional small trunks from veins surrounding the common bile duct insert at the superior or inferior aspects of the hepatic pedicle

Venous return from the common bile duct ramifies by the concomitant venous arcades accompanying the branches from the hepatic artery These venous collaterals empty into the splenic vein In about 20 per cent of cases certain venous tributaries join the superior mesenteric vein Free intrahepatic communications have been observed between portal and hepatic veins There have been no primary anastomoses observed between the hepatic artery and portal vein or between the hepatic artery and hepatic vein

The liver is drained by two major hepatic veins which enter the inferior vena cava beneath the diaphragm In about 15 per cent of cases the hepatic veins enter the vena cava through the right diaphragmatic crus These veins are 2 cm long are from 1 5 to 2 0 cm wide and usually empty separately into the cava although they are in juxtaposition In about 30 per cent of cases the right and left hepatic veins fuse into a single trunk No valves have been observed in the hepatic veins

The gallbladder is supplied by the cystic artery usually derived from the hepatic or right hepatic artery The artery is anterior to the common (or right hepatic) duct and superior to the cystic duct It may be paired It may be immediately adjacent to the cystic duct or separated from it by 1 5 cm The cystic vein is doubled and a branch usually accompanies the artery and the duct (fig 20)

2 Pancreas

a Arterial

Arterial blood supply to the pancreas is derived from the superior mesenteric artery and branches of the celiac axis The superior and inferior pancreatic arteries originate from the superior mesenteric artery, the anterior, superior, inferior and posterior pancreaticoduodenal arteries are derived from the gastroduodenal and/or the superior mesenteric arteries

The superior pancreatic artery may arise from the splenic artery It may come from any branch of the celiac axis and occasionally from the axis itself The artery supplies the superior border of the neck, body and tail of the pancreas

The inferior pancreatic artery usually arises from the superior mesenteric artery It may originate directly from the superior pancreatic artery, may be duplicated or may arise from the gastro-epiploic anastomosis It supplies the inferior border of the neck, body and tail of the pancreas

The posterior superior pancreaticoduodenal artery arises from the gastroduodenal artery It may supply the anterior aspect of the common bile duct It is the lateral arcade to the duodenal loop and sends multiple branches to the head of the pancreas the papilla of Vater and the duodenum It is usually paired with the anterior artery

The anterior superior pancreaticoduodenal artery is the terminal branch of the gastroduodenal artery It enters the duodenal sweep just past the bulb and sends branches inferiorly and medially to the left half of the arcade (Michels) It is located between the pancreas and the duodenum on the left (medial) surface and supplies the anterior aspect of the head of the pancreas

The anterior and posterior inferior pancreaticoduodenal arteries are branches of the superior mesenteric artery and its jejunal branch In about 30 per cent of cases one or another of these arteries may be derived from the celiac artery These vessels supply the intestinal tract primarily and usually are a single arcade from the transverse duodenum to the first jejunal loop Anastomosis through these vessels permits transection of duodenum and pancreas at almost any selected point

There are additional arteries to the pancreas which arise directly from the splenic artery

b Venous

The venous drainage of the pancreas and duodenum together with that for the adjacent common bile duct enters the portal vein by means of the splenic vein the superior or inferior mesenteric veins The head and neck

of the pancreas generally drain into the superior mesenteric vein. The body and tail of the pancreas drain superiorly into the splenic vein and inferiorly to the inferior mesenteric vein.

The anterior superior pancreaticoduodenal vein drains the anterior superior pancreatic head. It joins larger tributaries from the adjacent stomach and duodenum to enter the superior mesenteric vein.

The anterior inferior pancreaticoduodenal vein drains the uncinate process and adjacent head and neck of the pancreas. It joins larger tributaries from the terminal duodenum and jejunum to enter the superior mesenteric vein.

The posterior superior and inferior pancreaticoduodenal veins are behind (superior) and below (anterior) the common bile duct. They drain the head of the pancreas and the distal segment of the common bile duct including the papilla of Vater. These paired vessels may join the portal vein at the junction of splenic and superior mesenteric veins. Otherwise, they empty separately into the portal vein (postero-lateral) or superior mesenteric veins. In some cases (25 per cent) these vessels drain into the splenic and inferior pancreatic veins.

The inferior pancreatic vein is found along the posterior inferior border of the body and tail of the pancreas. It usually drains into the superior mesenteric vein and may have collateral branchings to the inferior mesenteric vein.

The splenic vein along the superior border of the pancreas drains the greater portion of its body and tail through numerous short tributaries.

The portal system is intimately related to the body and neck of the pancreas. The portal vein is formed by the junction of splenic and superior mesenteric veins beneath (posterior to) the neck of the pancreas. The superior mesenteric vein is usually formed from trunks paralleling the superior mesenteric artery. There are usually no branches into the portal vein between the superior aspect of the pancreas and the liver hilum except occasionally for one of the posterior pancreaticoduodenal veins.

II. Lymphatic Drainage of the Biliary Tract (Fig. 19)

Lymphatic drainage from the pancreato-biliary hepatic tissues eventually reaches the cisterna chyli. Between the renal veins and the hepatic veins are 1) the aortic glands which drain the kidney and adjacent area, posterior abdominal wall and peritoneal cavity, 2) the preaortic glands which surround the celiac axis, 3) the caval or lateral aortic glands at the entrance of the hepatic veins which drain a great portion of the liver—(a) inferior group: gallbladder, common bile duct, head of pancreas, (b) superior group: hepatic ducts and liver, 4) the superior mesenteric glands and 5) the pancreatico-lienal glands which drain the duodenum and pan

creas from pylorus, neck, body and tail of the superior and inferior pancreas and spleen

Lymphatic channels may communicate with the intercostal groups superiorly, and with the hypogastric or lumbar glands, inferiorly

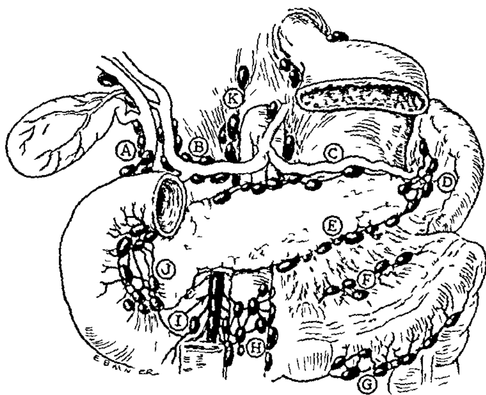


FIG 19 LYMPHATIC DRAINAGE FROM PANCREATO BILIARY TRACT

A cystic duct common duct nodes B hepatic artery nodes C splenic artery node D gastrosplenic and omental nodes E inferior pancreatic nodes F, mesocolic nodes G (greater) omental nodes H aortic nodes I external chain J pancreatic head node K preaortic nodes

I Innervation of the Biliary Tract

Extrinsic nerves enter the liver through the hepatoduodenal ligament, extend into the interlobar and interlobular septa among branches of the hepatic artery, portal vein and bile ducts. The hepatic ducts receive branches from the anterior hepatic plexus and from the cystic duct plexus. Liver lobules have visceral afferent nerves, unmyelinated in character, which follow the blood vessels. The bile canaliculi and capillaries have no smooth muscle and no nerves have been identified

Sympathetic and parasympathetic nerve fibers are derived from the celiac plexus to form anterior and posterior plexuses at the hepatic pedicle There are five major intercommunicating nerve plexuses 1) surrounding the hepatic artery and its branches 2) surrounding the hepatic vein and its venules 3) surrounding the portal vein and its ramifications 4) surrounding the intralobular ducts and 5) surrounding the interlobular ducts and major bile ducts

The anterior hepatic plexus follows the hepatic artery and its branches The anterior hepatic plexus originates on the anterior hepatic ducts and sends branches to the medial superior surface of the gallbladder as the medial cholecystic nerve The posterior plexus is found along the portal vein and common bile duct The posterior plexus sends branches as the lateral cholecystic nerve to the lateral surface of the common and cystic ducts and to the lateral and inferior surfaces of the gallbladder

The gallbladder has subserosal intramuscular and submucosal ganglion cells which form plexuses within each of these layers The common bile duct has subserosal as well as intramuscular ganglion cells The common bile duct is completely surrounded by a delicate network of nerve fibers

Moore has demonstrated experimentally that pain fibers supply the biliary tract along with the hepatic artery blood supply Stimulation of sympathetic fibers produces epigastric pain Vagus stimulation usually induces indigestion dyspepsia and vomiting

Bilateral vagotomy produces a decrease in myelinated fibers without affecting the sympathetic nerve distribution Extirpation of the celiac ganglia bilaterally will eliminate all sympathetic innervation In order to denervate the gallbladder and ducts it is necessary to do a bilateral vagotomy and a bilateral celiac ganglionectomy Even under such circumstances nerve fibers reach the biliary tract via phrenic components which reach the liver through the hepatic veins and peritoneal reflections

J Anomalies (cf Chapter I)

Biliary tract anomalies are reported frequently This does not indicate that they are common In fact all evidence available at present is that biliary tract anomalies are no more frequent than those in the genitourinary tract There are the same proportion of anomalous renal vessels or malposed ureters and exstrophies of the bladder as abnormalities of the hepatic vessels deformity of the common bile duct and a doubled or bipartate gallbladder

Certain errors in maturation deficiencies in structure multiplicities or atresias may be anomalous Anomalies have been reported in all portions of the hepato-pancreato-biliary tract

1 **Anomalies of the Liver**

a absence (incompatible with life)
b partial agenesis (atrophy of either lobe)
c reduplication
d transposition (*situs inversus*)
e associated with right diaphragmatic hernia (congenital)

2 **Anomalies in the Biliary Ducts** (excluding the gallbladder and the papilla of Vater)

a atresia of bile ducts, located
  (1) intralobar
  (2) at the hilum
  (3) right or left hepatic
  (4) common hepatic
  (5) common bile duct
  (6) transduodenal duct
  (7) segmental
b choledochal cyst
  (1) intrahepatic
  (2) replacing hepatic duct
  (3) replacing aberrant hepatic duct
  (4) representing reduplication of common bile duct
  (5) within the bile duct
c stricture
  (1) at porta hepatis involving junction of right and left hepatic ducts
  (2) of common hepatic duct
  (3) of common bile duct at area of union between cystic and common hepatic ducts
  (4) segmental
d accessory hepatic duct (usually on the right)
  (1) posterior to cystic artery
  (2) paired and parallel to common bile and hepatic duct
    (a) emptying into common bile duct
    (b) emptying into separate papilla
  (3) paired for short distance
    (a) surrounding right hepatic artery
    (b) surrounding cystic artery
  (4) entering cystic duct
  (5) entering gallbladder
  (6) entering junction between cystic duct and common hepatic duct
  (7) entering common hepatic duct distal to union of right and left hepatic ducts (fig 20)

e abnormal termination of hepatic duct (usually on the right)
 (1) right hepatic duct joins cystic duct and continues parallel to the left hepatic duct these reported as merging just prior to papilla (fig 21)
 (2) right hepatic duct terminates in gallbladder

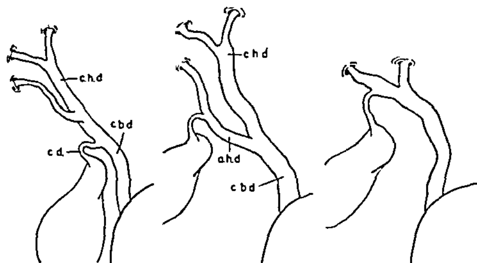


Fig 20 (*left*) Anomaly of Biliary Ducts

Accessory right hepatic duct joins the common hepatic duct (c h d) distal to the bifurcation for right and left hepatic ducts

Fig 21 (*center*) Anomaly of Biliary Ducts

Accessory (right) hepatic duct (a h d) joins the cystic duct which then joins the common hepatic duct (c h d) to form the common bile duct (c b d)

Fig 22 (*right*) Anomaly of Biliary Ducts

Cystic duct joins the right hepatic duct proximal to its union with the left hepatic duct By definition the common hepatic duct is absent in this case and the two hepatic ducts directly form the common bile duct

 (3) right hepatic duct originating at bifurcation of the common hepatic duct stops in the right lobe of the liver just adjacent to the gallbladder bed

f common hepatic duct
 (1) absent by definition (fig 22) because cystic duct terminates
  (a) into right hepatic duct
  (b) into left hepatic duct
  (c) into stomach
  (d) into duodenum

- (2) reduplication
  - (a) cystic duct merges with right hepatic duct the left hepatic duct and the right may join just proximal to the papilla there may be two papillae
  - (b) associated with reduplication of gallbladder and duplication of cystic duct
- (3) septum
  - (a) partial
  - (b) complete

g cystic duct

- (1) absent (cholecystocholedochal fistula)
- (2) duplicated
- (3) abnormal termination (right or left hepatic stomach or duodenum)
- (4) atresia
- (5) erratic terminus
  - (a) *anterior*
  - (b) posterior to left of common bile duct
  - (c) posterior to right of common bile duct (fig 23)

3 Anomalies of the Gallbladder

a absence (less than one per 1000)

b duplication (less than one per 4000)

- (1) with double cystic duct

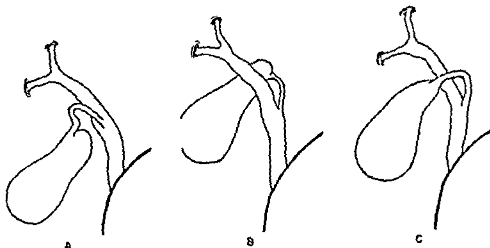


Fig 23 Erratic Termination to Cystic Duct

A On the anterior surface of the common duct
B Posteriorly to enter the left side of the common duct (cf Fig 42 B)
C Anteriorly to enter the left side of the common duct

(2) adjacent and with common peritoneal coat
(3) separated (one with the left lobe of liver and the left hepatic duct with total duplication of the common bile duct to its termination the other on the right)
(4) bilobed gallbladder with single cystic duct
(5) triloculation with double longitudinal septum and one or two cystic ducts (also associated with aberrant and accessory right hepatic duct)

c diverticula
d rudimentary
e abnormal external adhesive bands to duodenum colon kidney
f left sided (and with situs inversus)
g hour glass trabeculations
h intrahepatic
i floating
j cyst associated with atresia of the cystic duct

4 **Anomalies of the Portal Vein**

a absence
b duplication (actually represents failure in union between superior mesenteric and splenic veins)
c aneurysm
d varicosity or hemangioma

5 **Anomalies of Hepatic Veins**

a none reported except in association with agenesis of liver
(b differentiate from Budd Chiari syndrome)

6 **Anomalies of Hepatic Artery System (Fig 24)**

a accessory right hepatic artery
b abnormal position of hepatic artery
(1) crosses anterior to hepatic duct
(2) right hepatic artery anterior to right hepatic duct
(3) varicosity or aneurysm or tortuosity of hepatic or right hepatic artery in Calot's triangle

c accessory cystic artery from hepatic artery or from right hepatic artery
d cystic artery between hepatic ducts
e cystic artery behind common hepatic duct
f accessory cystic artery from left hepatic artery and posterior to left hepatic duct
(1) may have both cystic arteries from left hepatic artery

g accessory right hepatic artery
  (1) both from superior mesenteric artery behind common duct
  (2) one from hepatic trunk, one from superior mesenteric artery

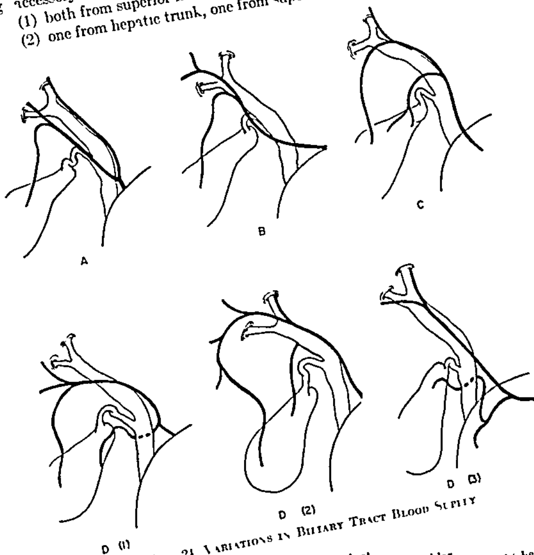


Fig 24 Variations in Biliary Tract Blood Supply

A Accessory right hepatic artery
B Common hepatic artery anterior to bile duct
C Two or three cystic artery branches reach the gall bladder
D The cystic artery may arise from 1) common hepatic trunk or right hepatic artery 2) simultaneously from multiple source arteries or 3) from the gastroduodenal artery (or its branches)

  (3) both from hepatic artery, one anterior, the other posterior to the common hepatic duct

h gastroduodenal artery may cross left border of (or entire) common bile duct, being mistaken for cystic or hepatic artery

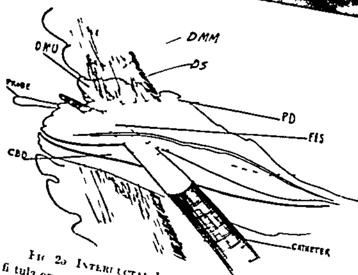


FIG 25 INTERDUCTAL FISTULA (CONGENITAL)

Congenital fistula originates beneath a valvule in the *pars intestinalis* of the bile duct A catheter passes through the fistula A metal probe is seen passing through the orifice for the pancreatic duct (Photograph by courtesy of *Am J Path*)

7 Anomalies of the Papilla of Vater

a agenesis, atresia
b *duplication associated with duplication of the common bile duct*
c malposition
  (1) in stomach
  (2) into pancreatic duct with pancreas
d atresia of pancreatic duct (fibrous or absence)
e diverticula of either or both ducts within the papilla
f intrapapillary choledochal pancreatic duct fistula (fig 2)
g associated with diverticulum of duodenum

8 Anomalies of the Duodenum

a persistent ventral mesentery
b *obstructive cystogastrocolic band (complete or partial)*
c stenosis
d atresia
e diverticula
f reduplication
g stricture (or intraluminal septa)
h cystic degeneration

9 Anomalies of the Pancreas

a atresia
b agenesis
c *heterotopia throughout gastrointestinal tract*
d cysts
e annular pancreas
f failure of fusion

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3

PHYSIOLOGY

A. Bile Flow

1. General

The common bile duct is a conduit serving to transport bile from the liver to the duodenum. This "pipeline" includes a "surge chamber" represented by the gallbladder which permits both storage and availability. The common bile duct has a "nozzle" at its termination which increases the flow of bile to jet stream velocity. These concepts conform to hydraulic principles. The basic physiology of the common bile duct pertains to transportation. An additional function is that the common bile duct may concentrate bile, particularly in the absence of the gallbladder.

2. The Sphincter

The termination to the common bile duct is a sphincter. Its function is independent of adjacent or contiguous duodenal musculature. Reactability of the sphincter, however, is influenced by variations in duodenal motility and tone as well as by changes in duodenal and intracholedochal pressures.

It has been assumed that the sphincter of Oddi may 1) permit the filling of the gallbladder, 2) prevent regurgitation of intestinal content into the bile duct, 3) regulate the discharge of bile into the duodenum, and 4) provide for erection of the papilla. A fifth function proposed by Archibald is segmental activity of the sphincter to partition flow selectively to and from the bile and pancreatic ducts. Not all of these are proven.

Bile enters the duodenum normally (Flman, Mann) at intervals of 1½ to 2¼ hours and only in small amounts during fasting. McMaster and Elman (1926) first indicated the primary mechanism for the expulsion of bile by the gallbladder to be a reciprocal relationship with the sphincter of Oddi. There are three factors (Bergh) in bile flow into the duodenum: 1) the tonus of the muscles around the lower common duct, 2) activity of the gallbladder, and 3) the pressure of bile secretion at the time of the ingestion of food. McMaster observed that bile flow occurred as an intermittent gush, and Kozoll and Necheles (1942) found the sphincter response to be independent of duodenal activity.

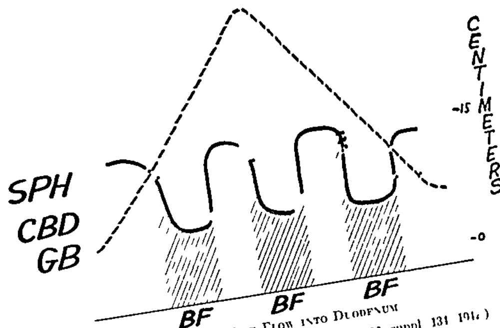


CHART I Bile Flow into Duodenum

(Adapted from Erik Hjorth Act Chirurg Scand 96 suppl 131 1947)

- - - pressure within gall bladder (GB)
—— presumed pressure at papillary sphincter (of Oddi) at the termination of common bile duct (SPH)
pressure of bile within common bile duct (CBD)
//// periods of bile flow from duct into duodenum (BF)

Bile is formed continuously Although the biliary ducts empty periodically the gall bladder is usually not ever completely empty

In the absence of adequate stimuli the papilla is closed During such intervals bile fills the ducts and the gallbladder When food is seen bile flow commences it ceases as food is eaten although at this time bile production is increased Digested food passing through the pylorus into the duodenum activates bile flow So also do various chemical neurogenic physical and hormonal stimuli affect relaxation of the sphincter increased intracholedochal pressure or both

This mechanism for bile flow is not under conscious control Pressures within the biliary tract vary with the rate of bile production and are modified by action at the papilla and within the gall bladder Normal bile flow into the duodenum occurs when duct pressure exceeds sphincter resistance

3 Bile Pressure

The secretion pressure of liver bile averages 32 cm (Herring Sutherland) The pressure in the gallbladder is usually 10 cm However in the fasting state pressure in the gallbladder may be elevated to 25 or 30 cm With the gallbladder intact the level of common duct pressure is usually not over 10 cm Pressure of 60 to 70 mm may cause bile flow from the common duct into the gallbladder Measurements in man of intraductal

pressure will usually measure sphincter resistance. The values have been obtained in patients who have been operated on because of biliary tract disease. In addition, such patients have had cholecystectomy and choledochostomy. These undoubtedly affect physiologic response of the duct and its sphincter. Further factors to be considered are constant addition of bile from the liver as well as its flow through the papilla.

Normal "base line" values in patients are those obtained 10 to 14 days postoperatively if the patient is asymptomatic. Normal sphincter resistance is between 9 and 23 cm. of saline (bile). Pressure greater than 12 to 15 cm. of bile normally is required to open the sphincter. Occasionally normal pressure may reach 35 cm. There are rhythmic contractions of the papilla observed every five or six seconds which last one to two seconds. The tone of the papilla normally varies. It is modified by (a) secretory pressure of the liver, (b) state of the gallbladder wall, (c) compression effect from the sphincter muscles at the lower end of the duct and (d) back pressure from within the duodenum. There is no change when vagus fibers are divided and sympathetic nerves around the hepatic vessels are intact.

Major control of bile flow at the papilla is by the circular muscle of the sphincter. When this is cut, there is no further control; all other effects are negligible.

Pain may be produced by dilatation of the extraduodenal common bile duct or by pressure or other stimuli at the termination of the duct. These may inaugurate additional reflexes in the arterial blood supply. In the abnormal duct, distention pain occurs at normal pressure levels. In the normal duct "distention pain" may not occur until the intraductal pressure or rate of flow is twice normal.

4. Factors Modifying Sphincter Resistance

Changes of 5 to 10 mm. in intraductal pressure occur during respiration. Greater changes (1.5 cm.) occur during coughing, laughing, vomiting, as a result of any temporary increase in the intra-abdominal pressure and following changes in body position. Even without stimuli, sphincter resistance may vary plus or minus 1 cm. of bile pressure.

After a calculus has been removed from the common bile duct, an irritable sphincter may persist for many weeks. Irritability presents abnormal responses to pressure stimuli by the papillary muscle. After continuous external drainage of the common bile duct, irritability disappears and the sphincter mechanism returns to normal.

Fats (such as egg yolks or cream) permit relaxation of sphincter tone. High protein concentrates (or amino acids) also do this. Carbohydrates in the duodenum or intravenously in high concentration have no effect on the sphincter. Large volumes of water or fluid containing electrolytes pass

ing through the pylorus or retained in the duodenum have no effect on the sphincter.

Drugs such as morphine, codeine, Pantopon and Dilaudid cause increased *tone in the sphincter of Oddi* and as a result greater intraductal pressure is produced. It takes higher pressure after those drugs to overcome sphincter tone. Amyl nitrite and nitroglycerin can decrease the tone of the sphincter and lower its resistance to intraductal pressure. Atropine, prostigmine, epinephrine, ephedrine, Demerol and ethyl alcohol have variable effects on sphincter relaxation. Magnesium sulfate in the duodenum will produce transient sphincter relaxation with an initial increase in tone. Phenobarbital can reduce sphincter tone. Tfocaine, Pontocaine and Nupercaine *applied locally will decrease* the amount of pressure required to open the sphincter. Novocain and procaine locally or intravenously usually have no effect.

Atropine, papaverine, calcium, Prostigmine or caffeine may permit the sphincter to open more easily. *Urecholine can provoke contractility of* the sphincter which is overcome by Bentyl, atropine and phenobarbital. *The effects of curare have not yet been fully determined. There is marked* individualization of response.

5. Factors Modifying Bile Flow

The effect of drugs on bile flow is evaluated on the basis of simultaneous effect on the gallbladder, on the sphincter of Oddi, on the common bile duct and on the duodenum. Some drugs stimulate parasympathetic nerves which contract the gallbladder and at the same time increase tone of the sphincter of Oddi. These, for example, include acetylcholine, muscarine, pilocarpine and physostigmine. Other drugs simultaneously relax the gallbladder and the sphincter. These include atropine, Banthine, scopolamine, ergotamine tartrate and caffeine. Atropine abolishes the tone of the sphincter of Oddi. It may increase sphincter activity when in combination with *epinephrine* and codeine. Nicotine increases sphincter and duodenal tone.

Frequently, morphine, codeine, Dilaudid or Pantopon in normal dosage increases intrabiliary duct pressure up to 200 or 300 mm. within several minutes. These effects can be antagonized by amyl nitrite or nitroglycerin. The effect of increased pressure in the duodenum or in the bile duct produced by narcotics may not be affected by atropine, *alcohol, ephedrine,* epinephrine, calcium chloride, papaverine, physostigmine, acetylcholine, ergotamine tartrate or caffeine.

Bile salts usually increase hepatic bile flow. Simultaneously gallbladder tone and common duct sphincter activity decrease. This may be due only to the fact that bile salts effect a marked dilution in bile. Magnesium

sulfate however causes increased flow of normal hepatic bile but the gall bladder is contracted and the sphincter open

6 Additional Mechanisms

There is a close association between physical chemical hormonal and pharmacological stimuli which affect the sphincter mechanism at the termination of the common bile duct It is probable that sphincter relaxation is coincident with stimulus to and contraction of the gallbladder

Bockus points out at least four trigger points 1) the junction of the gallbladder infundibulum 2) the neck of the gallbladder 3) the valves of Heister in the cystic duct and 4) sphincter muscles at the termination of the extraduodenal common bile duct These may act in unison or independently

The gallbladder will empty efficiently in the presence of fat in the duodenum Less efficient than fat but fully adequate as a stimulus is meat and its peptone proteins

Cholecystokinin extracted from duodenal and jejunal mucosa is produced in response to the presence of dilute hydrochloric acid in the duodenum Its composition is unknown although it is somewhat similar to secretin Quantitative measurement is standardized as one dog unit which intravenously increases intracholecystic pressure by 1 cm Cholecystokinin is present in blood in increased concentration after a fatty meal It has not been identified in blood in the fasting state It can be inactivated by a substance like a secretinase It is thought that the quantity of cholecystokinin determines the duration and extent of gallbladder contraction

Primary stimuli may be referred to the biliary ducts and gallbladder from within the brain stem by reflex from the gastrointestinal cardiac plexus Emotion and excitement such as fright pain and anxiety can cause increased sphincter resistance

The perception of food decreases sphincter resistance Eating increases the sphincter resistance until a half to one hour after eating when the resistance decreases again This coincides with rate of flow through the pylorus

During sleep abdominal muscles are relaxed Bile flow into the duodenum is decreased Bile formation in the liver however continues Therefore the lack of stimuli to open the sphincter permits the gallbladder to fill and bile to be concentrated This also occurs during pregnancy

Minor factors which may modify bile flow are (a) the elastic recoil of the gallbladder (b) the flushing effect of hepatic bile flow producing a siphonage (c) the milking effect of duodenal peristalsis (d) changes in posture

Mirizzi thought that peristalsis occurs in the common bile duct which

forces bile from the hepatic duct into the gallbladder He feels that active peristalsis in the common bile duct plus *relaxation at the sphincter permits* bile to enter the duodenum This has not been observed by others

In addition the contiguity of the pancreatic duct and the presence of secretin plus *cholecystokinin may halt bile flow but increase pancreatic* duct flow Since pancreatic fluid and bile both pass through the same sphincter, an imbalance may be mutually provoked

The intraductal secretory pressure of the pancreas is variable Maximum daily pressures average about 450 mm Pancreatic secretory pressure may be considered the resultant of two intraglandular processes namely, *secretion and resorption* A change in either process may cause a variation in the secretory pressure No correlation exists between rate of secretion and maximum secretory pressure

7 Physical Principles of Fluid Flow

The fundamental facts in physiology of the common bile duct are (a) the common bile duct acts as a conduit with a "surge chamber" in its line and a "nozzle" at its termination (b) the ducts transmit fluid in accordance with basic physical principles of hydrodynamics

Flow velocity and pressures in the common duct can be evaluated in accordance with laws of physics In dealing with 'fluid flow' and "velocity changes" variations in movement and the character of pressure waves may be measured [1]

Flow through the common duct is "unsteady" as opposed to "steady" When the distal end of the common duct is closed, the velocity of the bile

[1] For example, in the common bile duct the velocity of the pressure wave can be expressed as (Dougherty)

$$a = \sqrt{E_v \frac{g}{w}} \sqrt{\frac{1}{1 + \frac{E_v d}{E t}}}$$

where a = velocity of flow

E_v = 144 × 300 000 lbs /sq foot for water (bile)

g = acceleration of gravity (32 274 ft /sec /sec)

w = specific weight in lbs /ft

(square root of E_v times g/w is from 3300 to 4700 ft /sec)

d = diameter of duct (pipe)

E = linear tensile modulus of elasticity in wall (lbs /sq inch)

t = thickness of duct wall (pipe)

flow through the duct is reduced to zero As long as hepatic excretion of bile continues there is fluctuation in pressure which resembles a "water hammer" phenomenon The character of fluctuation depends upon the activity of the gallbladder and upon the secretory pressure of the liver bile

Fluid pressure increases as a convex curve which tends to flatten The sphincter opens instantly and closes slowly

When the sphincter is closed adjacent bile is at rest even though fluid is still entering the duct system at (initial) hepatic velocity and pressure Bile adjacent to the papilla is under increasing pressure The increased intraductal pressure is exerted circumferentially and is entirely independent of the duct length The duct wall is stretched

The terminal segment of the extraduodenal common bile duct may resist circumferential expansion by reason of adjacent (and frequently surrounding) pancreas Resultant forces against a weak point may result as a spontaneous perforation These are usually associated with structural deficiencies diverticula and retained calculi

Intraductal pressure forces are transmitted to the proximal extrahepatic ducts This force is a wave of unloading traversing the bile duct in retrograde manner as a water hammer phenomenon In the closed common duct the water hammer phenomenon is characterized by pressure waves which travel at high velocity with no or little physical movement of the contained bile

The water hammer pressure waves inaugurate movement of excess volumes of bile into the gallbladder surge chamber The gallbladder fills in conjunction with the closed papilla and fluctuations in common duct pressure become correspondingly slower and fewer and overall pressure subsides

If the gallbladder is full continued pressure waves can provoke pain and colic Expansion of the gallbladder increased further concentration of bile by loss of water and the opening of the sphincter at the papilla of course will lower intraductal pressure

The actual character of sphincter action at the papilla has not yet been positively defined The papilla does not act like the anal sphincter which usually needs associated voluntary control It does not act alone like the pyloric sphincter which responds to pressure gradients and is subject to additional physiologic control—nor does it act like the weak voluntary oral sphincter nor like the strong automatic pupillary sphincter The sphincter mechanism at the papilla of Vater has individual characteristics best compared to the nozzle on a fire hose which may be regulated by a thermostat

A nozzle is defined as a converging tube A tube is a short pipe

which may be straight, diverging or converging. A nozzle normally increases velocity of transmitted streams because the internal diameters decrease. In some cases the nozzle converges then diverges, presenting a short section of minimal area. Forced rotation of the stream due to changes in direction (twist) within the nozzle also increase velocity of transmitted fluids. In the bile duct rotation is produced in the stream of bile because of anatomical tortuosity of the duct.

B Liver

1 General

The liver is the body's largest gland providing *multiple complex and* specialized metabolic (secretory) and excretory functions. The liver weight is about 5 5 *per cent of an average adult yet it contains about 20 per cent of the normal circulating blood volume (800 cc). Total hepatic blood flow* averages 1500 cc per minute (1000 to 1850 cc per minute).

Approximately 75 per cent of hepatic blood flow originates in the portal system. About one fifth of the portal blood *flow is derived from the spleen*. This includes various products of blood cell destruction and some of the internal secretion of the pancreas. Portal blood from the stomach contains large amounts of sodium and sugar. Portal blood originating from the small bowel contains hormones, enzymes, electrolytes, chemicals, toxins, bacteria and bacterial products. Blood from the upper gastrointestinal tract goes to the right lobe of the liver, that from the spleen goes to the left portion of the liver.

During digestion and with increased body metabolism, portal blood flow increases. Normal portal vein pressure is 7 2 cm (saline). The hepatic *vein pressure* averages 2 1 cm. Pressure in the abdominal vena cava is approximately 1 8 cm.

Twenty five per cent of the hepatic blood supply is arterial. The hepatic artery *is not* large; the pressure in it is equivalent to that of any similar systemic artery and averages 120 mm Hg. The question is still unsettled as to whether the liver can survive loss of its arterial *flow*. *Usually infarction and necrosis* follow ligation of the common hepatic artery; deaths have been reported. The hepatic artery does not communicate with the

[*] Flow through the papilla resembles that through the nozzle on a pipe which reduces the discharge while giving the stream a higher jet velocity

$$k = \frac{1}{C^2} - 1$$

where k = *coefficient of loss*

C = coefficient of velocity

(1) storage of protein
(2) *protein deamination and urea synthesis* (conversion of proteins with production of specific dynamic action)
(3) plasma protein production
(4) destruction of uric acid

d storage of metals and vitamins

e detoxification

(1) detoxification by destruction (drugs), by conjugation (indoles and phenole) by selective retention and slow release (barbiturates) and by excretion into the bile

f erythropoiesis and blood coagulation

(1) *storage of blood*
(2) erythropoiesis and storage of antianemic principle
(3) maintenance of blood coagulation (production of fibrinogen prothrombin, heparin, bile salts and utilization of vitamin K)

2 Chologenesis

Volume of hepatic bile in the normal adult varies directly with the amount of food and may range from 500 to 1500 cc daily Ninety five to 97 per cent of bile is water Remaining constituents are

a pigment bilirubin (biliverdin)—1 0 gm per cent
b bile salts (tauro- and glyco cholates)—3 to 50 mg per cent
c bile acids (variable)
d lecithin (variable)
e cholesterol (12 to 26 mg per cent)
f inorganic salt (variable)
g muco and nucleo-proteins (variable)
h enzymes and antibodies (variable)
i calcium (2 to 10 mg per cent)

Bile pigments are derived by hydrolysis from hemoglobin and myoglobin The breakdown of the hemoglobin occurs in the reticuloendothelial system (Kupffer cells) in the spleen liver bone marrow and lymph nodes Bilirubin is a part either of hemobilirubin or cholebilirubin depending upon its chemical union with protein In the liver bilirubin is separated from its protein complex It then appears in the bile as sodium bilirubinate In the intestinal tract the bilirubin is reduced to stercobilin (urobilinogen) and this is oxidized to urobilin A large part of the urobilin is reabsorbed and returned to the liver where it is destroyed or re excreted Urobilin cannot appear in the urine unless it is absorbed from the intestinal tract following extrahepatic synthesis *Urobilin is not formed within the bile ducts* except in very rare instances of cholangitis associated with prolonged obstruction In addition to the reduction oxidation process which

converts bilirubin to urobilin there is also a direct oxidative process which forms biliverdin and bilicyanin from the bilirubin (Sackey) This latter process occurs frequently in the presence of active infection of the bile passages

Bile salts are produced by liver cells They are catalysts for absorption and utilization of fats and fat soluble vitamins

Cholesterol is found in blood in bile and in all tissues It is held in solution in the bile by bile salts The quantity of lecithin is also of importance in the ratio of cholesterol to bile salts Variations in lecithin content directly affect the amount of cholesterol held in solution by bile salts Decrease in lecithin causes crystallization of cholesterol unless bile salts concentration is increased Excretion of cholesterol in bile is increased in hyperthyroid and decreased in thyroidectomized rats By inference this may be similar in man

Inorganic salts and other breakdown products of liver detoxification processes are usually present in bile Antibiotics are found in bile Antibodies are present in bile Antigens such as bacteria and bacterial toxins as well as allergins can be identified It has been suggested that the agglutination (precipitation) reaction occurring between specific antigen and antibody may be essential (or catalytic) in formation of calculi (Sterling) Protein in minute amounts in the bile exists as the gamma globulin and as mucoprotein Some of the latter is derived from the glands in extrahepatic ducts Local formation of antibodies occurs (Ehrich)

Many factors influence the production and flow of bile

a Blood Circulation

Increase in liver blood flow increases bile formation Intravenous dehydrocholic acid increases the flow in the hepatic artery followed by an increase in bile volume This increase is due to added water As a result all constituents including antibiotics and bilirubin are diluted

Increase in portal blood flow increases the volume of bile and increases the percentage of pigment constituents but all other constituents are not proportionately altered

Occlusion (or ligation) of the hepatic artery will not be followed by cessation of bile production Occlusion of the portal vein alone will reduce bile production at least half

b Innervation

Splanchnicectomy may increase bile flow bilateral vagotomy may be followed by a decrease in bile flow These effects are due to the innervation of the hepatic artery and the portal vein

c Pharmacology

A "choleretic" is an agent which stimulates the liver to produce more bile by providing additional material used in bile production Among these are secretin, salicylic acid cinchophen pilocarpine acetylcholine, insulin choline and histamine

A "cholagogue" is a substance which stimulates bile flow by inaugurating stimuli which empty the gallbladder and bile ducts This is a "cholekinetic" function Food in increased volume will provide increased bile flow (vide infra) and magnesium sulfate will act so

Certain substances are both cholagogues and choleretics These include olive oil, egg yolk and certain fatty high cholesterol food substances

d Diet

Carbohydrates tend to diminish bile formation Proteins inaugurate and *maintain increased bile production Fats act to stimulate bile flow and also* produce specific effects by reason of associated contained protein

e Mental and Psychic

Rage suppresses bile production The individual *reaction* to pain also suppresses bile flow Vomiting is a strong stimulant to increased bile formation

f Hormonal

Cholecystokinin is liberated in duodenal and jejunal mucosa as a result of presence of fat It is presumed this substance can initiate bile flow and also stimulate contractility of the gallbladder For example fatty food introduced into the duodenum of a denervated biliary tract will produce both contraction of the gallbladder and choleresis This also occurs experimentally when blood obtained at the height of digestion is given intravenously (*Fasting blood will not inaugurate choleresis nor evacuation* of the gallbladder) The same reaction occurs with cholecystokinin extracted *from the duodenal or jejunal mucosa*

3 Metabolism and Other Hepatic Functions

a Glycogen and Fat Metabolism

The hepatic cell assists in regulation and maintenance of normal blood sugar levels This is accomplished by converting food into carbohydrate *(glycogen) and by keeping sufficient glycogen available for rapid mobilization* when required for body needs It has been shown that proteins and fats may be converted into carbohydrates by the liver and that when certain enzymes and catalysts are functioning the glycogen may be con

verted to essential amino acids The exact mechanism for this remarkable chemistry has been identified but not clarified

When the liver cell has been damaged by disease by hypoxia by chemicals or toxins or by aberrations in its blood supply the first rapid indication of malfunction is the disappearance of liver cell glycogen Many years ago Ravdin indicated that low fat high protein and adequate carbohydrate intakes assist adequate glycogenolysis and fat metabolism Based upon his work is the present day evaluation of adequate hepatic cell protection using 1) high protein and carbohydrate intake 2) adequate oxygenation and 3) use of such enzymes as lipocaic and such essential amino acids as methionine and choline with the administration of lecithin

Ketone body production occurs during glycogenolysis That ketones are not properly neutralized may indicate further functional defects in hepatic or pancreatic function

Severe liver necrosis is often characterized by a marked hypoglycemia A rapid decrease frequently occurs The glycogen has to be replaced by 10 per cent glucose intravenously at an average rate of 120 cc per hour

b Protein Metabolism

The liver stores protein It converts protein by a deamination process so that specific dynamic action provides calories The formation of urea is also a detoxification mechanism Decrease in blood urea nitrogen may indicate the onset of hepatic cell failure The liver is the major source for urea nitrogen

The liver destroys uric acid the end product of purine metabolism It converts portions of available hemoglobin and other breakdown products to albumin It utilizes the globulin fraction and makes it available for antibody formation in the reticuloendothelial system lymph nodes and elsewhere Fibrinogen is among other fractions formed by liver cells

Excess protein can be adequately metabolized by the liver Failure in function of the kidney is evidenced by blood stream accumulation of such end products of liver metabolism as urea indole and phenol

c Storage

Liver cells retain metallic and other elements as iron copper molybdenum zinc magnesium vitamins A D and K and a factor which catalytically opposes primary pernicious anemia The Kupffer cells can retain such elements as thorium for years

d Detoxification

The hepatic and Kupffer (reticuloendothelial) cells cooperate in detoxification Bromsulfalein excretion tests this phase of hepatic function If

detoxification is not adequate, general liver function may be failing Such drugs as cincophen, arsenicals, strychnine, alkaloids and the barbiturates are destroyed The indoles and phenoles are conjugated in the liver prior to renal excretion Some of these may be selectively retained depending upon transcellular concentrations and may be slowly excreted into the bile Salicylate and many antibiotics are also selectively filtered They are not changed, they are retained and slowly excreted Contrast media used for radiological visualization of the biliary tract are (modified slightly and) excreted over a 24 hour (oral) or 6 hour period (intravenous)

e Erythropoiesis and Blood Coagulability

Deficient hepatic cellular function contributes to both macrocytic and microcytic anemias *The liver will tend to replace red blood cells prior to* adding to protein partitions if there is a choice available to it Frequently, hypoalbuminemia and its consequent reversal of albumin globulin ratio are an early indication of hepatic insufficiency The protein partitions including fibrinogen are made almost entirely in liver cells

Heparin and prothrombin together with their catalysts and antienzymes are all formed in the liver Failure of storage or manufacture of prothrombin is an early indication of hepatic cell incompetency *Heparin is a constant,* its deficiencies are rare Together with other facets of erythropoiesis, the liver will store and can produce a major amount of antibodies These relate mostly to the lymphatic tissues Hepatic venous blood and bile are rich in both agglutinins and precipitins Hepatic bile and serum have equivalent *concentrations of antibodies* The gallbladder concentrates antibody in almost the same ratio as bile pigment (Sterling)

The liver is a frequent site for shock states *for allergic manifestations in*cluding the Schwartzman phenomenon Liver function is affected by and influences many phases of sensitization and similar states

C Physiology of the Gallbladder

Heister (1728) and MacLurg (1772) first differentiated hepatic bile from gallbladder bile by the increased viscidity of the latter Rous and McMaster *(1920) and Ravdin Johnston Riegel and their group (1932)* first presented a firm basis for understanding the precise function the gallbladder played with reference to the hepatic bile both *normally and in disease*

In fact prior to 1931 there had been doubt that bile left the gallbladder through the cystic duct there were many (Sweet and *Heidenhain*) who felt that bile entering the gallbladder through the cystic duct was discharged through lymphatics and blood vessels Ivy, Graham Elman and McMaster, Boyden and finally Ravdin s group demonstrated conclusively that the gallbladder evacuated concentrated bile through the cystic duct

Although certain constituents of the bile are found in lymphatics and blood vessels these do not include bile pigment or cholesterol

The cystic duct offers very little impediment to flow in and out of the gallbladder Ordinarily bile will enter the cystic duct at pressures between 10 and 80 mm

Regardless of the physiologic state of the gallbladder whether water is being slowly or rapidly absorbed or whether the gallbladder is emptying or filling the bile in the gallbladder tends to reach isomolar concentration with that of serum This does not apply to pigment mucin or cholesterol

Bile pigment may be found in hepatic bile at 0 38 to 1 5 gm per cent (Graham and Whipple) It may be concentrated by the gallbladder up to eight or nine times

Mucin of irregular content has a concentration co-efficient of 8 85 in the gallbladder

Cholesterol in hepatic bile ranges from 12 to 26 mg per cent The gall bladder may concentrate it an average of 7 5 times

Water is rapidly absorbed

Chloride in hepatic bile usually ranges from 76 to 110 m Eq /l It can rapidly be absorbed

Calcium in hepatic bile is found in concentrations of 1 9 to 10.2 m Eq /l It is not absorbed normally However its concentration is not increased and if present in excess of isomolar concentration it may be absorbed

Bile salt in hepatic bile ranges from 3 3 to 52 6 m Eq /l Bile salts may be absorbed through the gallbladder wall

Energy for selective concentration of bile constituents by the epithelium is evidently provided by the oxidative breakdown of carbohydrates and lipids In this chain of reactions inhibition of succinic-dehydrogenase participates in depressing inspissation and viscidity (Mustakallio) In late pregnancy high production of estrogens inhibits the succinic hydrogenase system Estrogen reverses the inhibition of increased viscidity normally present There is then an increased inspissation This has been demonstrated to occur in the liver cells and gallbladder epithelium in the mouse

It is of significance not only because it emphasizes the importance of interaction and interdependency of the various enzyme systems but also because it stresses again the intimacy among cholesterol hormones and vitamins

In the damaged gallbladder changes in absorption and dilution phenomena occur Bile salts and calcium concentration are decreased partly by a dilution phenomenon In addition chloride concentration pH and CO are increased possibly because absorption by gallbladder epithelium is prohibited Of all these factors decrease in bile salt concentration is most significant because under such conditions the supersaturation of cholesterol

(held in solution by the bile salts) may cause crystallization of the cholesterol and provoke cholelithiasis

Many, many theories have been advanced regarding the etiology of cholelithiasis. In the 1500's Paracelsus presented a theory that gall stones *were due to impurities in the system.* Thudicum in 1863 thought that a putrefactive process was responsible. Naunyn felt that stasis and infection together were the cause. *Aschoff and Bacmeister attributed the mechanism* to stasis and hypercholesteremia. Lichtwitz explained the stone formation on a physiochemical phenomenon. Sterling in 1936 (*confirmed by Marteinson* in 1939 and others) presented a theory in which the agglutination (antigen antibody) reaction was a trigger mechanism *compounding alterations* in the bile salt, cholesterol and lecithin ratio.

No theory has been positively proven at this time.

Cholelithiasis frequently follows pregnancy. Cholecystitis may originate during early months of pregnancy. Pregnancy and other factors such as congestion, hypercholesterolemia, thyroid ovarian pituitary imbalance, vitamin deficiency, calcium imbalance and residual or complicating inflammatory or degenerative diseases all play a part in lithiasis (Fallon and many others).

Based upon the capacity of the gallbladder to concentrate and alter bile constituents, two other factors have been noted during pregnancy. First, cholesterol concentration in (hepatic and gallbladder) bile increases during pregnancy. Secondly, bile salt concentration is lower. These *per se* may establish suitable conditions for stone formation in the biliary tract.

D. Duodenum

The duodenum contains the same digestive glands (including Brunner's) *which are present throughout the intestinal tube.* It incorporates the exits for the two powerful digestive enzyme groups, namely, bile and pancreatic juices. Its functions are digestive as well as to transmit chyme.

1. Digestive

Intestinal glands throughout the mucosa produce a variable secretion which usually consists chiefly of water and salts and has few, if any, digestive ferments. The secretion may be slimy *if much mucus is present.* It is usually alkaline (7.0 to 8.5 pH) because of sodium carbonate and bicarbonate. Enterokinase is present *to activate trypsin. Lipase and amyl*ase are formed in traces by the intestinal mucosa. Certain peptidases like erepsin and nuclease, arginase and phosphotadase are present. Specific ferments for sucrose, maltose and lactose may be isolated.

Brunner's glands' functions have not been specifically identified. They are presumed to form cholecystokinin and secretin in addition to quantities of mucus and carbonates.

Duodenal gland secretion responds to both nervous and hormonal stimuli Mechanical stimuli in the passage or retention of food residues can cause excitation of the glands with resultant increase in liquid content and *provocation of duodenal peristalsis It is believed that the sympathetic* nerves inhibit secretion Vagus effect is on the whole inconclusively demonstrated

Secretin acid content from the stomach pancreatic juice and increased pressure in the duodenum all act as stimulants to duodenal gland secretion In addition in the duodenal (and upper jejunal) mucosa there is cholecystokinin This appears only subsequent to the passage of food through the duodenum and is greatest in concentration when the largest amount of food is being digested by the stomach

The physiologic mechanisms for discharge of bile and pancreatic juice have been discussed elsewhere

2 Movement of Duodenal Content

The third and fourth portions of the duodenum like the remainder of the small intestine react to the presence of a food bolus by peristalsis including mass movement Antiperistalsis is a normal activity in the second and third portions of the duodenum

The first portion of the duodenum (duodenal cap) is closely coordinated with mechanisms of the pyloric antrum and pyloric canal The second portion of the duodenum receives bile and pancreatic juices although it exercises minimal if any control thereupon

The pyloric sphincter is the key to the physiology of both the antrum and the duodenal cap Contraction of the pyloric antrum is associated with relaxation of but followed by contraction in the duodenal cap Pyloric sphincter contraction occurs about five times per minute and usually begins when the duodenum is relaxed Increased duodenal pressure will not only initiate increased peristalsis but will also stimulate the pyloric sphincter to close With the duodenum empty the pyloric sphincter becomes sensitive to stimuli from the antrum and will relax in response to pressure waves initiated within the stomach More frequent than peristalsis through the duodenal cap is the fact that this area will empty by overflow into the second portion of the duodenum

F Pancreas

Pancreatic cells constantly form both external and internal secretions

1 External Secretion

The circulation in blood of secretin formed within the duodenal mucosa will proportionately increase the rate of pancreatic cell activity Vagus and

sympathetic nerve fibers play important roles in ferment secretion It is felt however, that the water and salt actually representing the major volume and effectiveness of the pancreatic juice, are regulated by secretin

Atropine can decrease the volume and lipase content of pancreatic juice without affecting the amylase content Banthine and ephedrine are similar Alcohol orally or intravenously produces no change or a decrease in pancreatic juice volume Cortisone has no effect Prostigmine, on the other hand will increase the secreted volume and will increase ferment content

Ferments in the pancreatic juice include trypsinogen, chymotrypsinogen diastase lipase, amylase maltase, lactase and rennin Their concentration in the normal 1200 to 1500 cc of daily pancreatic juice is variable (chapter 7)

According to Howard (who studied a case of pancreatic fistula) the normal external secretion has a pH of 8 5 and specific gravity of 1 015 There is 0 8 gm of protein per 100 cc albumin is 0 5 and globulin 0 3 gm per cent Chlorides vary between 35 and 97 m Eq /l and the bicarbonate concentration between 30 and 74 m Eq /l , sodium is 140 m Eq /l , potassium is 5 0 m Eq /l and the calcium is from 1 0 to 5 0 mg per cent Amylase is normally below 300 units Lipase ranges between 300 and 500 units

Some investigators have identified "lipocaic" as relating to fat metabolism and pertinently responsible for preventing fatty infiltration of the liver This has not been sufficiently confirmed

Total pancreatectomy is compatible with life insofar as the external secretion is concerned (chapter 5)

2 Internal Secretion

Although "insulin" was identified in 1922 by Banting and Best as the internal secretion of the islands of Langerhans, we are not fully apprised today concerning its precise and complex mode of action It certainly is not required in calculated quantity following total pancreatectomy The diabetic state may require 60 or more units of insulin whereas the postpancreatectomized patient rarely can use more than 20 units

Total pancreatectomy is feasible through proper replacement of the internal glandular secretion In presence of pre existing hyperinsulinism due to an islet cell tumor the status of the patient following its excision may be precarious since the normal pancreatic function may have been depressed After a few days however normal control is re established

I Common Bile Duct Physiology—Postoperatively

One means for physiologic evaluation of the common bile duct following cholecystectomy is by pressure volume study through the choledochostomy

In the event that direct communication with the duct is not available, studies may be done by gastroduodenal intubation and radiography (Cholografin).

Following cholecystectomy the residual volume of the normal bile duct can be 5 cc. Variations from 0 to 8 cc are common. This varies because of factors such as food stimuli, state of tonus of the duodenum, content of the duodenum, intraperitoneal pressure and the patient's state of mind. Residual volumes greater than 9 cc, however, are associated with some obstruction at the termination of the common bile duct.

The normal resting pressure of the common bile duct ranges up to 8 cm. When the residual pressure is greater than 10 cm, a calculus may be present or there may be edema or traumatized redundant mucosa at the papilla.

As fluid is injected into the duct, intracholedochal pressure rises in a ratio of 1 or 1½ to one until a maximal pressure of 15 to 18 cm is present following the instillation of 10 to 15 cc. At this level the patient may complain of moderate discomfort and a sense of abdominal fullness. This is relieved as intracholedochal pressure decreases.

The duct volume normally decreases at a rate of 2 to 3 cc per minute. The normal duct returns very quickly to its original volume and pressure (chapter 15).

The value of tests of intracholedochal pressures and volumes has to be carefully scrutinized. Inadequate position of the tube or other technical errors are obvious as a rapid loss of fluid around the tube or as a varying character to the discomfort produced by injected fluid or by markedly irregular response to the injected fluid.

It must be determined that measurements are actually being taken of the fluid content of the common bile duct. It must be known that there is no leakage through or around the "T" tube or that the tube was not kinked. It must then be decided that the fluid filled the common bile duct and/or the hepatic radicles to capacity. If the enlarged radicles are filled prior to the opening of the sphincter of Oddi, there can be greater duct capacity. This is particularly true in chronic calculous obstruction to the common bile duct wherein the papilla of Vater has an altered reactivity threshold.

An evaluation of pain reactions must be made in regard to the distensibility of the common bile and hepatic duct walls as compared to the reactions at the sphincter of Oddi (chapter 15).

G. Physiologic Changes in the Biliary Tract Produced by Other Systems

On many occasions exploration of the biliary tract reveals dilatation of the bile duct—without any evidence of inflammatory disease of the gall

gallbladder or ducts, no abnormalities at the papilla or pancreas, and apparently without cause. Search through all adjacent structures and tissues may also fail. Under such circumstances myriad theories appertain.

Cholecystitis with or without lithiasis may be present. Stricture or spasm at the papilla may exist. Exact and specific data are required because should certain maneuvers be done in the bile ducts or gallbladder the patient may be propelled along continuing invalidism rather than be relieved of symptoms.

Changes occur in the biliary tract secondary to functional stimuli and organic disease elsewhere (particularly in the gastrointestinal tract). Edema of the pancreas can occlude the common bile duct. A sensitization phenomenon or inflammation may inaugurate a hepatitis together with a pericholangitis. Hiatal hernia or achalasia in the course of digestive disturbances which ensue may fully disorganize biliary tract function. Cholecystitis and sphincter dyskinesia are often found with constipation and as a sequel to repeated catharsis. Pleuritis and pneumonia, cardiac insufficiency and coronary artery disease, cirrhosis and ascites, appendicitis and peptic ulcer and primary and metastatic malignancy all affect bile duct function. It should be noted that the hepatic pedicle and the second portion of the duodenum are frequent sites of metastases.

Secondary changes in the biliary tract then may not be relieved until the primary disease is adequately treated. This particularly applies to physiologic changes in the biliary tract associated with angina with coronary artery insufficiency and in myocardial infarction. The two systems are mutually imbalancing, clinical and physiologic evaluation is often impossible.

In addition, surgical procedures involving other systems may produce physiologic alterations in the biliary tract. For example, following gastrectomy technical difficulties may result in occlusion to the common duct. More important, however, is the postgastrectomy status (except in the gastroduodenostomy) wherein biliary tract function is altered by the absence of food from the duodenum. Even in the gastroduodenostomy the absence of a pyloric sphincter eliminates a very important source for normal biliary tract stimuli. Re-establishment of physiologic balance is accomplished by an increased formation of cholecystokinin and by an alteration in papilla and gallbladder function. It should be stated that occasional episodes of the "dumping syndrome" may be due to minor alterations in bile flow.

Furthermore, vagotomy for peptic ulcer may increase the volume content of the gallbladder and bile ducts. This leads to stasis and may perhaps inaugurate biliary tract disease.

In addition, sympathectomy done for hypertensive disease can neutral-

ize the appearance of symptoms which might identify pancreatitis or cholelithiasis until such times as serious complications overwhelm the patient

Recognition of the normal response by the bile ducts to the presence of cardiac gastrointestinal and renal disease should be carefully differentiated from primary physiologic changes in or disease of the biliary tract

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4

PATHOLOGY

Tissue alteration observed at the operating table is often different from that seen at postmortem examinations. In the biliary tract inflammation, adhesions, dilatation of ducts, calculi, tumor, deformity of visceral contours and relationships are often the only evidence to explain symptoms. At operation liver and pancreas may grossly be abnormal but (fig 26) even frozen section microscopy may not clarify the diagnosis. Yet every effort is expended to make a positive diagnosis despite difficulties. The presence of normal tissue seen under the microscope at the time of surgery does not exclude the existence of infection nor does presence of infection exclude malignancy.

At operation gross and microscopic examination of tissue may differentiate cholangitis from hepatitis, cholestasis from cirrhosis, carcinoma from fatty degeneration, and hepatoma or hemangioma from metastatic malignancy (figs 26a, 26b). Frequent errors occur particularly in decision concerning the character of a pancreatic lesion which is palpably stony hard.

It is difficult, often impossible, to differentiate by frozen tissue study the normal, atrophic, inflamed or malignant disease of the pancreas. However, it is essential to procure as much information as possible at the time of operation so that proper definitive decision for therapy be made if at all possible.

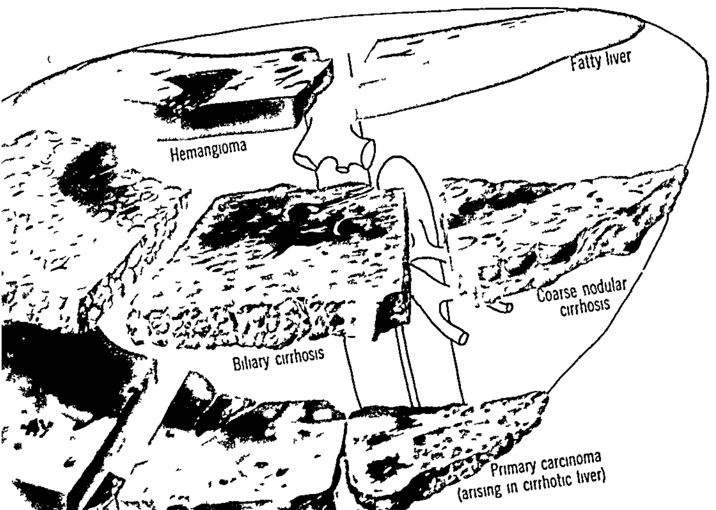
Disease of the bile ducts is manifest in the ducts, in adjacent tissues, in the liver and pancreas. Portal cirrhosis may secondarily involve the biliary tract. Pancreatic disease, such as calcinosis or infection, may be accompanied by hepatobiliary manifestations. In these instances biliary calculi which may be present do not necessarily account for the patient's symptoms.

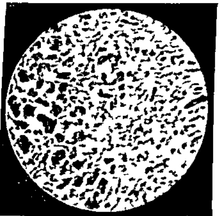
(Certain aspects of bile duct pathology are considered in Chapters 5, 6, 7 and 15.)

A. Inflammation of the Bile Ducts

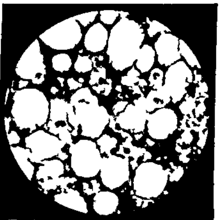
1. Infection

Acute, subacute or chronic infection of the common bile duct may involve the mucosa (cholangitis) or the entire wall of the duct in addition to the


Fatty liver
Hemangioma
Coarse nodular cirrhosis
Biliary cirrhosis
Primary carcinoma (arising in cirrhotic liver)



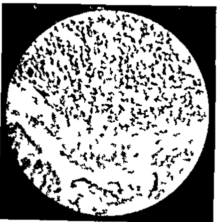
Cirrhosis



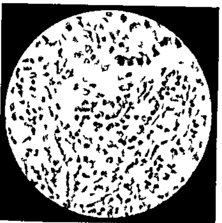
Fatty liver



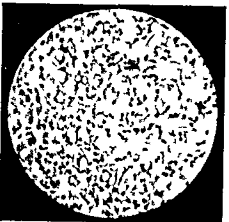
Carcinoma



Acute hepatitis



Cholangitis



Amyloidosis

FIG 26 GROSS AND MICROSCOPIC PATHOLOGY IN THE LIVER (COURTESY CHAS PFIZER & CO INC)

A Gross findings *Hemangioma* A common benign lesion which often simulates metastasis It may be red, *orange or brown* *Fatty liver* is large pale yellowish and greasy *Cirrhosis* of many varieties is manifest as irregular nodules of regenerating liver distorted by surrounding pale atrophic or scar tissue The liver in biliary cirrhosis is firm green and frequently nodular the extrahepatic bile ducts are usually dilated *Abscess* in liver is usually adjacent to severe gallbladder infection may be single or multiple Hepatitis may be diffuse as from viral infection or localized surrounding gallbladder inflammation Pylephlebitis and abscess formation can occur through venous routes *Congestion* is present in the bile duct due to hepatic vein dilatation enlarged pulsating liver in cardiac decompensation is dusky violaceous *Parasitic cysts* may be deep or superficial as rounded dense brown or grey globules of variable size

B Microscopic findings *Cirrhosis* exhibits periportal fibrosis and irregular areas of hepatic cellular regeneration In bile cirrhosis the cholangioles and bile capillaries *are dilated In portal cirrhosis bile capillaries may be increased in number* (regeneration) but are not usually dilated *Fatty liver* may appear as degeneration or infiltration and is diffuse *Carcinoma* is usually characterized by pleomorphism anaplastic cells with mitosis *Hepatitis* presents varying degrees of periportal necrosis *Cholangitis* evidences periductal necrosis and polymorphonuclear cellular infiltration *Amyloid* liver manifests an abnormal perivascular substance associated with hepatic cell atrophy



FIG 27 CHOLANGITIS

Microscopy in cholangitic necrosis found at autopsy following chronic choledocholithiasis reveals much necrosis and inflammatory cellular infiltration

periductal tissues (choledochitis)[1] Inflammation may also involve adjacent tissue and peritoneal reflections The presence of an impacted calculus may inaugurate local inflammation of the common duct wall

Infections of the bile ducts may originate from the bile liver duodenum appendix intestinal tract and from adjacent abscess such as caused by peptic ulcer perforation The usual bacterial flora of the intestinal tract are the common invaders of the bile ducts Streptococcus and colon bacillus are most often found

Cholangitis can ascend to the intrahepatic canaliculi Progressive involvement of portal areas is often fatal in patients who have chronic cholangitis associated with choledocholithiasis (fig 27) Polymorphonuclear cellular infiltration frequently accompanies obstructive biliary cirrhosis Atrophy and fibrosis are often present in primary cirrhosis whereas necrosis more often accompanies infection However both cirrhotic and inflammatory states frequently co exist

Occasionally solitary or multiple abscesses may occur in the liver Per

[1] Differentiation between cholangitis and choledochitis is valuable to the surgeon Cholangitis demands adequate drainage of bile through the duct Choledochitis requires adequate drainage of periductal tissues

foration of these may produce one or another variety of subhepatic abscess (chapter 15)

2 Vascular Lesions

The blood supply to the common bile duct is segmental, marginal with adequate intramural anastomoses and with many collaterals Endarteritis and arteriosclerosis do not often appear in the vessels of the bile ducts Varicosities or hemangiomata of the common bile duct are seen frequently Aneurysms uncommonly are present There are several clinical reports of fatal aneurysms of the cystic artery and hepatic artery Portal vein angiomata have been reported

Ligation of the hepatic artery may occlude the hepatic blood supply Collateral supply to the liver and to the common bile duct, however, is excellent The right gastric artery often sends anastomoses to the liver hilum beyond the usual site of ligature by way of the hepatoduodenal ligament The pancreatoduodenal and gastroduodenal vessels provide additional anastomoses to the common duct Accordingly, ischemic gangrene of the common bile duct is virtually unknown

Ligation of the right, left or main hepatic artery can produce segmental or subtotal hepatic ischemic necrosis The degree of vascular damage produced in the liver varies individually Animal research studies are not fully illuminating This ischemic necrosis which is uncommon should not be confused with infarcts of Zahn which are not infarcts but areas of hyperemia associated with hepatic cell atrophy without necrosis Fatalities have been reported associated with segmental ischemia and necrosis of the liver

3 Infection and Vascular Occlusion

Complicating vascular occlusion of the liver and bile ducts is the presence of infection due to *Welch bacillus anaerobic streptococci* and other bacteria The secondary infection in the liver and bile passages is often abated following the use of broad spectrum and type specific antibiotics

Localized vascular occlusion may occur secondarily to infection of the gallbladder or bile duct It may be due to an impacted calculus in a mucosal fold of the gallbladder or common duct An opening may occur in the necrotic area and the gallbladder or duct will be perforated

4 Gas Bacillus Infection

Acute pneumocholecystitis due to an infection with gas forming bacteria is manifest by the sudden appearance of gas in the gallbladder and biliary ducts *Cl welchii B coli* Streptococci (both aerobic and anaerobic) and *Staphylococcus albus* have been found (Bell Brown and Lenhardt)

Extensive studies by many investigators have shown that the gastro-

intestinal tract and the gallbladder are the normal habitat of the above mentioned organisms in patients Local necrosis is a prerequisite for the establishment of a gas bacillus infection

The presence of gas in the gallbladder alone does not make the diagnosis Gas from the intestinal tract may enter the gallbladder through an internal biliary fistula

5 Calculi and Infection

Increased intracholedochal pressure usually accompanies biliary tract infection In addition if a calculus is present at one site continuously a diverticulum perforation and a bile fistula may occur

In the extraduodenal common bile duct the lumen is wide and the wall is thin In the transpapillary or transduodenal portion of the common bile duct the lumen is decreased and the wall is thicker A stone usually is impacted proximal to the thickened portions of the common bile duct papilla and the narrow filamentous portion of its lumen It may also impinge on a valvule in the papilla There is a tendency for a stone to erode through the thinner tissues of the extraduodenal common bile duct towards the pancreas and its duct or towards the duodenal wall (fig 28)

When there is a suspicion of abnormality in the biliary ducts (at autopsy) the common bile duct and its adjacent tissues should be placed in formalin prior to dissection Diverticula or fistulous tracts (1 mm in diameter and 1 cm long) are easily destroyed in fresh tissues by enzyme activity and dissection Abnormal channels such as diverticula between the common bile duct and the pancreas have been found in certain patients with pancreatitis Diverticula from the common bile duct through the pancreas and into the pancreatic duct can permit bile to enter the pancreas Epithelial metaplasia

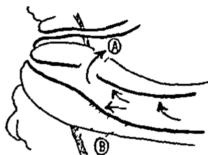


FIG 28 ROUTE FOR CALCULUS THROUGH PAPILLA

The normal route for calculi less than 3 mm diameter is through the duct If the calculus is larger than 4 mm (in response to increased duct pressure against the mass of the papilla) the calculus may erode (a) towards the pancreas and its duct or (b) into the duodenum (Courtesy of *Am J Path*)

FIG 29 DIVERTICULA OF BILE DUCT WITH ADJACENT PANCREATIC NECROSIS
Diverticula (DI) from the common bile duct enter an area of pancreatic necrosis (PAN NECR) The common channel (CC) for bile and pancreatic ducts is distal to the diverticula (Courtesy of *Am J Path*)

as described by Rich and Duff is seen in close proximity to diverticula *Edema necrosis and fibrosis are also seen in the pancreas (fig 29) These* may obstruct the common bile duct Diverticula of the common bile duct may be empty or may contain calculi (figs 30a, 30b) They may connect (fistula) to the common duct, duodenum or pancreas (fig 6)

Common duct stones may produce tremendous organic change or none Patients over 80 years of age with stones in the common bile duct may have adequate bile flow and only slight dilatation of the duct On the other hand a young patient with recent symptoms may have great dilatation of the common bile duct severe cholangitis and hepatic failure due to one or two small stones in the common bile duct The size of the stone and its duration in the common bile duct are not of primary importance The most serious effect of a stone in the common bile duct is to obstruct the flow of bile Once that has occurred then inflammation suppression of bile formation duct perforation jaundice or hepatorenal failure can supervene

Calculi and infection in the bile ducts are usually present together in the biliary tract Infection may exist without a calculus However cellular inflammatory reaction is almost invariably found when choledocholithiasis is present

The common duct stone is nearly always derived from the gallbladder

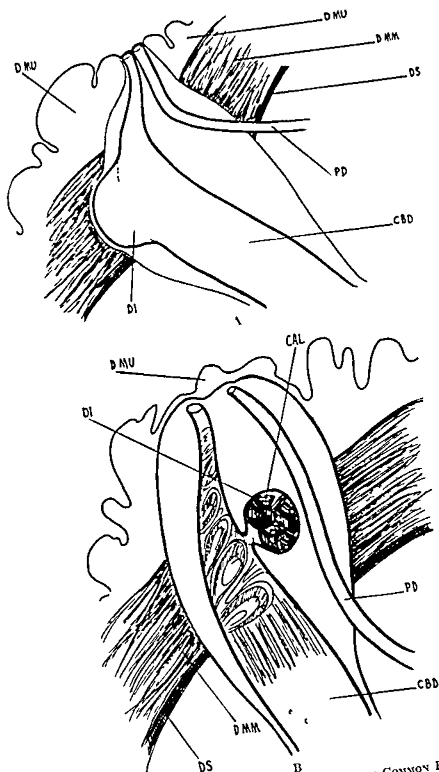


FIG 30 DIVERTICULA OF PAPILLARY PORTION OF COMMON BILE DUCT
A Without calculi diverticulum (DI) impinges toward duodenal wall
B With calculi diverticulum containing several small stones penetrating toward the pancreatic duct (Courtesy of *Am J Path*)

Yet, it is neither inconceivable nor disproven that stones may form in the *biliary ducts* Many factors undoubtedly participate in the formation of calculi Of these, stasis of flow is most *important* Auxillary factors such as *supersaturation of precipitable* substances are present Cholesterol calcium and bile pigment are excreted as solutions from the liver Changes in their *state depend upon the water content of bile, the activity* of enzyme systems such as cholinesterase the quantity of catalyst such as lecithin, the *specific function of chemical concentration* such as bile salts and acids and other factors

A most important step in stone formation is the presence of bacteria The frequent occurrence of cholelithiasis in typhoid fever is an example *The mechanism of stone formation in the presence of infection has been presented as a result of an antigen antibody reaction* (Sterling) The agglutinate *(bacterial bodies) or precipitate (toxins) is as a nidus within the duct* gallbladder or epithelial tissues whereupon materials in supersaturated *solution may then be deposited*

This is of importance in patients who have had choledochostomy Mucus production and epithelial desquamation *which accompany cholangitis may* in themselves provide a fertile field for the *formation of stones in the duct* even years after the biliary ducts had been demonstrated to be clear

Whether local or parenteral use of antibiotics or antisera will prevent *recurrent stone formation is not known It is not known which patients* have the capacity to form calculi nor under what conditions, nor at this time is there any satisfactory prophylactic

Findings at operation or by the pathologist in patients who have choledocholithiasis may be minimal However, there is usually found (a) common duct dilatation greater than 1 cm in diameter and (b) thickened common duct wall with mucosal erosions, edema and inflammatory exudate with fibrosis of the periductal tissues

Hepatic duct calcinosis has been noted occasionally in the absence of cholelithiasis In the usual case however, a calculus in an hepatic bile duct has been driven there by the distal obstruction to bile flow

The gallstone is usually of greater specific gravity than bile That it is not always so depends upon the water content of the bile and the constituents and porosity of the stone Radiographically calculi may be differentiated from air bubbles or mucous shreds by behavior and movement (chapter 8)

The first reported case of gall stones in an infant was recorded by Lieutaud in 1767 He found *calculi in the gallbladder and ducts of a 2 day* old infant at autopsy Since that time about 90 cases in infancy have been reported *(Potter) Why it occurs is a matter of conjecture* Cross and Ladd have suggested congenital hemolytic anemia as a basis for the formation of

stones in infancy Other conditions are believed favorable to the formation of stones such as biliary stasis infection and cholesterosis (Kellogg)

Pericholedochitis or abscess in the foramen of Winslow can compress the common duct However the outer coats of the common bile duct are extremely resistant to infection The outer coat of the common duct acts as a barrier to infection Tissue reaction subsequently may produce stenosis of the duct and thereby obstruct bile flow

6 Biliary Cirrhosis

Extensive periductal inflammation may be followed by fibrosis both within the liver and also in the hilar structures constituting the hepatic pedicle This not only affects a decrease in arterial supply but also can establish extrahepatic block to the portal vein and inaugurate irreversible portal hypertension This is a frequent sequel to stricture of the common bile duct wherein surgical trauma to the duct and its surrounding structures has added inflammation of the hepatic pedicle to the deleterious effects of bile duct obstruction Inflammatory reaction at the liver hilum may be fibrinous (Boyd)

Sequellae of hypersplenism and splenic vein thrombosis occur with purpura anemia and splenomegaly as significant findings These may also occur following simple cholecystectomy, that they are coincidental cannot be disproven Splenectomy and splenorenal shunt in these patients is a formidable surgical program and can be considered only in patients with reasonably good liver function and in whom there is a justifiable life expectancy

As a result of any biliary tract disease, obstruction to the portal vein and the splenic vein can occur I have seen a patient following cholecystectomy and one following repeated reconstructive procedures on the common bile duct who had splenomegaly In the first case purpura was associated with the splenic enlargement in the other portal hypertension caused fatal gastric hemorrhage In similar cases splenectomy has been done Both sequellae are on a mechanical rather than on a degenerative or an inflammatory basis

B Trauma

Trauma to the biliary tract may follow blunt force in automobile accidents or falls Trauma to the biliary duct may be due to weapons of violence and direct puncture injury may be produced

Injury to the common bile duct can be produced at surgery because of 1) adhesions 2) anatomical anomalies or 3) inadvertent manipulation In addition there may be deformity due to 4) pre existing disease Such trauma may occasionally not be noted at the time of surgery

There are several types of traumatic manifestations 1) the postchole

cystectomy syndrome, 2) biliary fistula, 3) bile duct stricture 4) bile peritonitis, 5) bile ascites (E, vide infra) and 6) obstructive jaundice (F, vide infra)

1 Effects of Surgery

The postcholecystectomy syndrome cannot exist in the absence of surgery on the biliary tract After surgery on the biliary tract the following may be present (a) residual calculi in the biliary ducts, (b) new calculi formed around debris (c) shrunken remnant of the cystic duct with contained calculi (d) dilated remnant of cystic duct with pseudocholecyst (e) cholangitis and choledochitis (f) stricture (g) adhesions, (h) duodenitis and papillitis (i) cholangiolitis and hepatitis (j) pancreatocholedochal inflammation (k) abnormal reflux between bile and pancreas or pancreatic ducts, (l) compression of bile duct by diseased adjacent tissue, (m) spasm inflammation, hypertrophy or tumor of the papilla of Vater and the sphincter of Oddi, (n) anomalies, (o) ventral hernia, (p) neuroma of the amputated cystic duct, (q) chronic infection in the liver bed and (r) foreign body cysts surrounding stones dislodged from the biliary tract (Cf also chapter 15)

2 Bile Fistula

Common bile duct fistulae may communicate with the pancreas, duodenum, gallbladder colon, stomach or peritoneal cavity or externally (cutaneously) A bile duct to-intestine fistula may precede intestinal obstruction due to gallstones in the intestinal tract However, intestinal obstruction or 'gallstone ileus" is more common when the stone comes from the gallbladder through a cholecysto-enteric fistula In such cases intestinal obstruction is most frequently at the terminal ileum and ileocecal valve In the presence of previous abdominal or pelvic surgery gall tone obstruction may occur at a site of bowel adhesion in the jejunum or ileum Other sites for gallstone obstruction to the bowel have been reported at the sigmoid colon following cholecysto-colic fistula and at the ligament of Treitz

A biliary tract enteric canal fistula may persist In such cases air may be visualized in the hepatic radicles (fig 15) and cholangitis is present Patients may have repeated episodes of gallstone ileus

Pathological findings observed in internal biliary fistula include pericholedochitis and abscess with thickening of the tissues surrounding the common duct However abscess may be minimal and the fistula is frequently re epithelialized A fistula between the termination of the common bile duct and the duodenum may not be identified

Perforation between gallbladder and common bile duct is not uncommon following necrosis of the gallbladder in the course of acute cholecystitis (Strelinger)

3 Stricture

There are two common sites for traumatic stricture of the common bile duct These are at the papilla and at the junction of the common hepatic and cystic ducts Inflammatory stricture of the common bile duct is found in the pancreatic portion of the extraduodenal common bile duct

The diagnosis of stricture or spasm is made too frequently at the papillary portion of the common bile duct The normal transpapillary common bile duct may be 2 5 cm long and from 0 5 to 2 mm in diameter The common bile duct is dilated on the hepatic side of the stricture The transpapillary portion of the common bile duct is normal when the lumen of the common bile duct is 1 cm or less in diameter Under such conditions there is no stricture spasm nor disease present at the termination of the duct The bile duct dilates to 1 cm or larger in the presence of stricture of the extra duodenal portion of the common bile duct The intrahepatic ducts are larger than 5 mm in diameter

C Neoplasms

Benign and malignant neoplasms of the bile tract are being found with increasing frequency (with the aid of the operative cholangiogram) Differential diagnosis at the operating table between stone inflammation, neoplasm and congenital anomaly in the bile ducts is very difficult Ordinarily *the presence of a benign papilloma of the papilla of Vater is not observed* until autopsy It can be missed even then if there is no suspicion These benign structures may be found only after thorough cross section study of the papilla

1 Benign Lesions

An intraductal papilloma of the common bile duct originates from duct epithelium A pseudo' papilloma may be formed as a tissue reaction to the presence of stone This granulation tissue is to be differentiated from neoplasm Although neoplasm may be found together with stone in the common bile duct the two are not definitely interrelated

Benign liver cell tumors include solitary and multiple hemangioma hepatoma and hamartomas Hamartomas of the liver are composed of liver cells bile ducts, blood vessels and the supporting connective tissue It is a developmental error where normal cells are in disarray The hamartoma is frequently well encapsulated and may be removed entirely

Benign cysts of the common duct can cause intermittent jaundice Warren and Pararo reported a thick walled cyst measuring 3 7 cm in length with a short pedicle attached to the wall of the common bile duct at the junction of the right and left hepatic ducts The cyst almost occluded

the lumen of the common bile duct The gallbladder was dilated In the mid portion of the liver there were numerous small and large thick walled cysts which were filled with clear fluid or with yellowish green material The cysts for the most part were intercommunicating and the entire region constituted a mass approximately 7 cm in diameter The common bile duct contained no stones

Chu recorded the type and frequency of the 55 benign tumors of the common duct as follows polyp, 21, adenoma, 18, fibroma 3 neuroma 3 lipoma 3 granuloma 2, melanoma 1, and carcinoid 1 Growth th growths are small firm and well circumscribed tumors within the walls c the bile ducts (fig 31) The cystadenoma on the other hand is a larg smooth round or oval tumor and contains thick, viscid fluid The cyst w is composed of fibrous tissue in which blood vessels and acini may be foun

2 Cholelithiasis and Carcinoma (Gallbladder)

From 60 to 95 per cent of patients with carcinoma of the gallblad have cholelithiasis There is no such correlation known between chole cholithiasis and tumor of the bile ducts

Calculi contain about 75 per cent cholesterol This is of interest bec



Fig 31 Benign Adenoma Biliary Tract

of possible carcinogenic capacity inherent in the abnormal metabolism of a sterol or bile acid to an aromatic hydrocarbon such as methylcholanthrene. Fieser has studied relationships among aromatic hydrocarbons, endocrine steroids and various oxidation or disproportionation reactions with reference to cholesterol. It is well known that the cholesterol in bile is the same as present in plasma and as in such high concentration in the red blood cell membrane. Cholesterol is converted to pregnanediol which is an equivalent of progesterone. In addition, there is adequate evidence that cholesterol is also a precursor to vitamin D_3. However, all efforts to the present have not yet succeeded to demonstrate the *in vivo* formation of a carcinogen from a steroid or to detect such a substance with relation to cholesterol.

A derivative of cholic acid, however, is identified in bile. This substance, the only known possible carcinogen, is methylcholanthrene.

Rolleston and McNee cite Maximilian de Stoll of Vienna as being the first (1771) to describe carcinoma of the gallbladder. It was considered to be rare until Graham (1928) reported that carcinoma of the gallbladder comprised 8 per cent of all malignant tumors. Since that report other studies have been published placing the incidence of this condition at about 4 per cent of all cancers. Necropsy reports from 23 authors totaling 206,098 autopsies revealed 908 instances of carcinoma of the gallbladder or an overall incidence of 0.43 per cent. Cooper found in a study of 2941 autopsies made at the New York Hospital during the 20 year period 1915 to 1935, 18 instances of carcinoma of the gallbladder. This is an incidence of 0.61 per cent. Kirshbaum and Kozoll carefully reviewed 13,330 postmortem examinations and found carcinoma of the gallbladder in 0.41 per cent of the cases. Detailed reviews by various clinics show the usual ratio of carcinoma of the gallbladder to carcinoma of the bile ducts to be about 6:5. More than 6000 deaths each year in the United States are due to carcinoma of the gallbladder.

Most malignant tumors of the gallbladder are a type of infiltrating adenocarcinoma. This invades all layers of the gallbladder, extends into the liver and produces widespread metastases. The next most common type, the papillary adenocarcinoma, is not as malignant. This grows more slowly, is more bulky and metastasizes later. It may be found unexpectedly at operation for acute or subacute cholecystitis. Squamous cell carcinoma is also found. Characteristically, biliary tract tumors are desmoplastic.

3. Malignant Tumors

Neoplasms of the liver include the malignant hepatoma 75 per cent, the bile duct cell carcinoma (or cholangioma) 20 per cent, and others such as endothelioma and hemangioblastoma 5 per cent. Metastases can be found

in from 25 to 65 per cent of cases in bones, lungs and adjacent tissue. By far the greatest number of neoplasms in the liver are metastatic from other organs.

Carcinoma is found in the common bile and in the hepatic ducts, the cystic duct and the papilla of Vater. Renshaw credits Durand Fardel with reporting the first case of cancer of the common duct in 1840.

Tumor is not common in the cystic duct; many investigators are not convinced that it exists. Many of these lesions may have infiltrated from the adjacent common hepatic duct or from the gallbladder. Tumors of the common bile duct including the papilla comprise over 70 *per cent of those* in the ducts (table 7).

Adenocarcinoma is the most common tumor of the hepatic ducts. Carcinoma and papillary carcinoma are also found. It is found in more males

TABLE 7. DISTRIBUTION OF NEOPLASMS IN THE BILIARY TRACT
(Kirshbaum and Kozell)

Gallbladder		218
Extrahepatic ducts		90
Cystic duct	10	
Common hepatic duct	16	
Common bile duct	35	
Papilla of Vater	29	
Unclassified (ducts)		110
		418

than females. It is characterized by metastases through lymphatics, through duct wall and by local infiltration. Late metastases occur into the liver.

Infiltrating and medullary adenocarcinomas compose the major type of tumors in this group. The infiltrating type is frequent in the common bile duct. Papillary adenocarcinoma is most frequent at the papilla of Vater. It is not unusual to find a localized primary tumor of the extrahepatic system.

In general the carcinomas of the extrahepatic bile ducts follow several distinct patterns of growth. In one instance there may be a diffuse infiltrating growth involving long segments of the duct. This may resemble a thick hard cord with obliteration of the duct lumen (fig. 32). Another type of tumor is similar in character to the "napkin ring" intestinal tract carcinoma. This is a malignant stricture of the bile duct and produces marked duct dilatation on the hepatic side of the neoplasm (fig. 33). The third type of tumor is the fleshy and fungating growth which may project into the duct lumen (fig. 34).

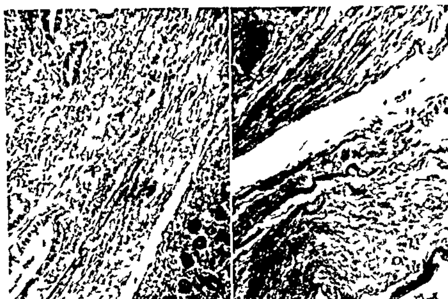


FIG 32 CARCINOMA AT PAPILLA
(left), adjacent to duodenal wall (right) adjacent to tran papillary pancreatic duct

Histologically these lesions may be markedly undifferentiated (anaplastic) or differentiated with cells varying from cuboidal to columnar

Marshall found metastases in only 25 per cent of cases Cole reported four cases of carcinoma of the papilla with no extension However Kirshbaum in 62 cases of extrahepatic duct carcinoma found metastases in 80 per cent

Spread by intramural perineural and perivascular extension occurs together with intraductal extension more frequently than lymphatic extension Lymph node metastases involve those around the head of the pancreas Other lymph nodes involved are in the cystic duct, the porta hepatis and the pre aortic groups Later metastases occur as peritoneal seedings to the adrenal the intestinal mesentery the gallbladder the liver and the lung

It is difficult to determine whether tumor at the papilla of Vater originates from the papilla itself pancreas pancreatic duct, bile duct or duodenum Bile duct carcinoma usually spreads along perineural lymphatics towards the liver hilum and the celiac plexus By contrast carcinoma at the papilla remains localized for great periods and spreads first by contiguity up the duct wall then through blood vessels (fig 33)

Tumors of the pancreatic duct tend to spread through the pancreas its capsule, its lymphatics and eventually hematogenously Tumors of the

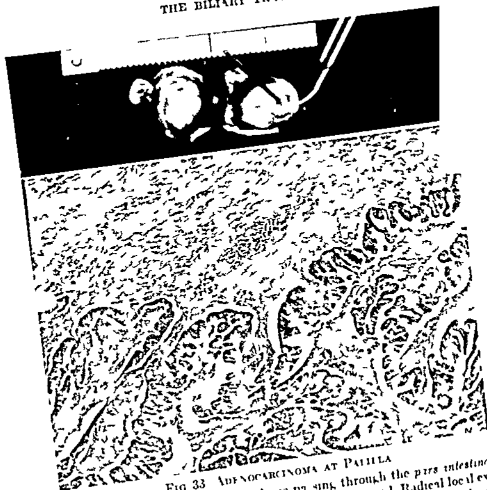


FIG 33 ADENOCARCINOMA AT PAPILLA

Originating within the papilla Probe is passing through the *pars intestinalis* of the bile duct Pancreatic duct is intact although compressed Radical local excision of the papilla was done (cf Fig 80) Microscopy reveals differentiated adenocarcinoma Four year survival to date

pancreas (fig 35) spread throughout the body of the pancreas, to adjacent or contiguous structure and then by lymphatic and hematogenous routes

Identification of the tumor origin can guide and may permit logical extirpation Frozen section study may permit classification Diagnosis can not always be made it should be attempted If the tumor for example can be classified as originating in the bile duct this tumor can be treated by local excision of the adjacent area in the pancreas and duodenum and by massive resection of the biliary ducts up to the hepatic hilum As much bile duct tissue is removed as is possible

Carcinoma of the pancreas is about three times more frequent in the head than in the body or tail One carcinoma of the body of the pancreas is found to three of the head of pancreas in 1000 autopsies The greatest incidence of pancreatic tumor is in the sixth decade



Fig 34 Adenocarcinoma of Bile Duct

Microscopy indicates extensive intraductal extension of tumor originating in bile duct epithelium Fatal 10 months



Fig 35 Carcinoma of Pancreas

Infiltrating lesion caused obstructive jaundice Pancreato duodenectomy was done Survival without clinical metastasis three years to date

Except for the presence of jaundice after the common duct is compressed signs and symptoms do not vary significantly with the location of the pan creatic neoplasm within the pancreas Metastases occur most commonly in the regional nodes, liver, omentum gastrointestinal tract adrenals and bones

The tumor which originates in the pancreas, however, should be treate by (total) pancreatectomy and partial gastrectomy together with duoden ectomy In this case the biliary ducts may not have to be removed

Carcinoma of the duodenum is exceedingly rare It has been the subje of several excellent treatises notably those of Hoffman and Pack (19 and Dixon (1946)

Tumor originating in the duodenum near the papilla may be confu with one originating in the bile or pancreatic ducts Usually the duod



Fig 36 Adenocarcinoma of Duodenum

Malignant tissue (on left) is seen microscopically adjacent to normal duodenal mucosa Pancreatoduodenectomy provided comfort for eight years In 1951 the pa tient required a colostomy to relieve intestinal obstruction caused by rectal shelf metastasis which invaded the sigmoid colon

carcinoma is a flat ulceration It may infiltrate papillary tissues However, it metastasizes early and marked lymphadenopathy is frequent in the adjacent mesenteries and omenta (fig 19)

These carcinomas are similar in all respects to those which occur in other portions of the gastrointestinal tract (figs 36a 36b) The adenocarcinoma may occur as one of three types (a) a scirrhous annular tumor with over production of fibrous tissue and a tendency toward constriction and obstruction, (b) a bulky polypoid intraluminal mass prone to ulceration, partial obstruction and hemorrhage (c) colloid carcinoma with mucoid degeneration

All types of these tumors may obstruct the bile ducts depending upon their location If sufficient time elapses they will invade surrounding structures including the pancreas and mesenteric vessels The scirrhous tumors tend to grow slowly, and in these invasion may occur late This type of tumor should have a high subtotal gastrectomy a total duodenectomy, a partial pancreatectomy and a distal segmental choledochectomy with cholecystectomy (if indicated)

D Congenital Lesions (Cf Chapter 2)

There are two categories of congenital lesions involving the bile ducts First are those associated with atresia or other malformation of the bile ducts which may be fatal in early infancy (fig 37) Second are those malformations of the bile ducts which may not obstruct bile flow

There are certain structural variations These involve the number of the ducts their location their course and their surroundings particularly the blood vessels and nerves

From the surgical point of view the hepatic bile ducts the termination for the bile ducts within the duodenum and the relationship between the common bile duct and portal vein are constant Other relationships of the ducts are variable For example the hepatic artery, cystic artery and branches thereof may alter their courses to overlie any quadrant of the common duct (fig 13) The hepatic ducts may branch into two or three major ducts (fig 14) The common duct may be duplicated The cystic duct may be long or short The gallbladder may be rotated upon itself and displaced

Mentzer found two hepatic ducts one of them entering the gallbladder and continuing by way of the cystic duct into the duodenum the second bypassing the gallbladder entirely emptying directly into the duodenum

There is another case reported by Schachner in which the excretory apparatus of the liver was arranged so that the bile passed directly through the gallbladder on its way to the intestine The gallbladder was small Its wall had three openings in addition to the cystic duct which was also the

common bile duct, there was also a main hepatic duct and a small orifice *which led to a cyst of a hepatic duct*

In the case of an acquired cholecystohepatic duct fistula, a large stone goes from the gallbladder into the common duct The absence of the cystic duct is due undoubtedly to erosion of the cystic duct by the stone (Behrend and Cullen) *In three reported cases all had pre existing chronic cholecystitis* obstructive jaundice and an artificial fistula caused by erosion of the stone In no case of this character was it possible to identify the cystic duct and at operation the common duct may be mistaken for the cystic duct A case, reported by Strelinger, had hypersplenism following a cholecystohepatic fistula Mirizzi has reported cases of a choledochal fistula cholecystohepatic fistula, cholecystohepaticocholedochal fistula and a combined cholecystohepatocholedochal and cholecystoduodenal fistula

A choledochus cyst or other abnormal dilatation may occur in the common duct These appear when the gallbladder is absent It is unusual except by size and pressure, for the choledochus cyst to produce symptoms in infancy However it is not uncommon between five and ten years of

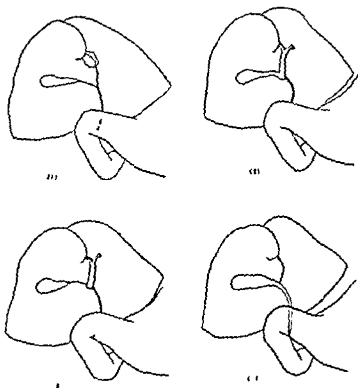


Fig 7 Biliary Tract Atresias

1 hepatic cystic and common bile ducts 2 common bile duct 3 gallbladder common bile and cystic ducts 4 common hepatic duct

age. There also may be co-existant vascular anomalies affecting the liver and spleen.

It is to be noted that between the third and fourth months of fetal life the hepatopancreatic tube which had been open in the very early fetal life becomes solid, filled with epithelial tissue. After the fourth month the liver ducts move towards the duodenum. Both the intestinal tract and the ducts then open again. It is at this stage when atresia of various segments of the bile ducts occurs.

Atresia, obliteration or block of the bile ducts may occur within any of the liver ducts, at the junction of the cystic duct and common hepatic ducts, at the common bile duct or at the termination of the duct at the papilla of Vater. The gallbladder may be atretic or may be fully developed. The fully developed gallbladder may have an atretic cystic duct. The size and content of the gallbladder usually provide a clue to the anatomical state. If atresia is above the junction of the cystic and common ducts it is usual for the gallbladder to be small and non functioning. If the atresia is in the common duct or distal to it, the gallbladder is enlarged and functioning (fig. 37).

Hepatic duct atresia has not been considered operable. However, if an intrahepatic bile duct be identified, adequate anastomosis may be performed. An extrahepatic atresia does not mean that the liver ducts are likewise narrow. Bile formed by liver cells can accumulate in the liver ducts which dilate. These may be made accessible by hepatotomy.

Congenital anomalies occur at the termination of the common bile duct. Among these are: 1) absence of the pancreatic duct, 2) absence of sphincter muscle, 3) interductal fistula (fig. 25) and 4) diverticula.

The papilla may normally be found from 5 to 15 cm. from the pylorus — usually about 7 cm. Location anywhere in the descending limb of the duodenum is normal. Rarely, the papilla may be found at the pylorus or in the adjacent stomach.

There may be two ducts entering separately through separate papillae. There may be a common duct draining the left hepatic duct which has an accessory common duct formed from the cystic duct and draining the right hepatic duct, each emptying separately into the duodenum. Other anomalies include a single common hepatic duct and a double common bile duct with these opening into the duodenum and into the stomach.

Anomalies in position and components of the papilla are less common than suspected. The normal (*pars intestinalis*) course of the common bile duct through the papilla is thin and tortuous and may be 3 to 25 mm. long. The normal papillary structure of the sphincter muscle may vary in thickness from 3 to 12 mm. To be anomalous or abnormal in size the papilla and ducts must be larger or smaller than these measurements.

Diverticula of the common bile duct in the supraduodenal portion may be related to the early stage in the formation of a choledochus cyst. The termination of the common bile duct may be within a large diverticulum of the duodenum.

Duodenal diverticula often are juxtapapillary. The duodenal sleeve through which the papilla enters is also adjacent to the double arcade of vessels. Both anatomical facts permit formation of the congenital diverticulum of the duodenum. Diverticula of the duodenum may include the papilla or may be adjacent to it. The diverticulum may provoke adjacent inflammation and the peridiverticulitis may cause pancreatitis. On the other hand, duodenotomy (with and without sphincterotomy) is not followed by diverticulum formation. Diverticula of the duodenum imitate cholangitis, hepatitis, biliary dyskinesia. They may be significant in the postcholecystectomy syndrome, in biliary cirrhosis and chronic pancreatitis.

In patients with duplication of the duodenum (with or without cysts) the biliary duct system may be duplicated. Heterotopic pancreas may appear in the gallbladder or bile ducts.

I Jaundice (Cf Also Chapter 5 and Plate One)

There are several states of jaundice which must be differentiated: first, congenital anomalies, second, inflammatory disease of the liver and ducts, third, hemolytic disease, fourth, neoplasm, and fifth, toxic factors.

The hemolytic group is most common in infancy. However, jaundice due to hemolysis may subside to be followed by a secondary obstructive jaundice due to inspissation of thickened bile or mucus plugs. This can be relieved by such cholagogues as magnesium sulfate, sodium sulfate, bile salts or antispasmodics. It is the rare patient with this syndrome in whom surgical clearing of the bile ducts is necessary. This is not usually fatal.

Inflammatory causes for jaundice include sepsis, syphilis and hepatitis. Systemic infection with hemolytic streptococcus, colon bacillus or Welch bacillus may have associated hemolysis. In this type of jaundice there is usually no evidence of obstruction. Patients with congenital syphilis who have hepatic enlargement may or may not have acholic stools. History, serology and x ray examination of bones may present evidence of syphilis.

Hepatitis of viral infectious origin may be increasing in incidence. In 20 per cent of such patients with hepatitis obstructive jaundice can occur due to cholangiolitis or inspissation of bile content. This can be confusing clinically. Atresias of the common bile duct have been discussed in the preceding section concerning congenital anomalies.

Absence of bile (acholia) in the intestinal tract is usually due to obstruction to the flow of bile through the common bile duct. In such cases there is

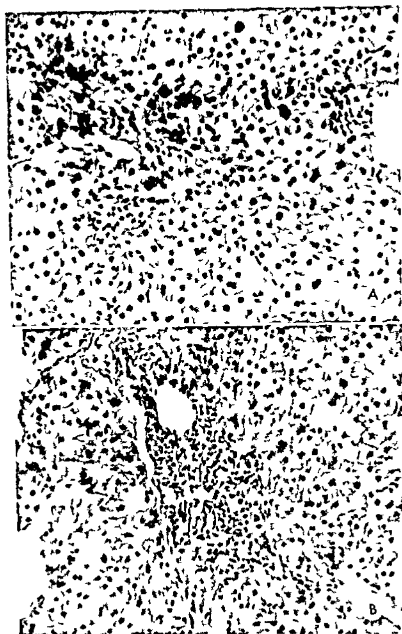


FIG 38 CHOLANGIOLITIC HEPATITIS

Cf also Fig 94

A Cholangiolitis is manifest by swelling of bile capillary epithelium the canaliculi are dilated debris is present within the lumen and bile pigment appears to be inspissated (often confused with other iron containing pigment) Hepatic cells are often replaced by or infiltrated with fat Eosinophilic infiltration is present in periportal areas

B The hepatitis phase of cholangiolitis is early manifest by findings in a with superimposed balloon swelling (with nucleolar changes) of the hepatic cells Necrosis as in cholangitis (cf Fig 27) may occur

produced first an increase in size and capacity of the bile ducts and gall bladder. Concurrently, there is an elevation of intracholedochal pressure. A balance is achieved between the secretory (and excretory) functions of the liver and the duct pressure in the duct system. The hepatic cell may not function when intraductal pressure is greater than 300 mm. of bile (Cantarow).

However, there are several factors in such circumstances which abet hepatic cellular functions. Since the biliary duct walls are elastic, the volume contained in the system can expand. This decreases the contained back pressure. Thereafter certain constituents of the bile trapped within the duct system may be absorbed. Usually this occurs when the gallbladder is intact and functioning normally. As a result of both dilatation and absorption, therefore, the observable effects of biliary duct obstruction may be delayed.

Bile then may regurgitate (or reflux) into the perilobular spaces in the liver (Rich and Duff). Here safety factors have been overcome and hyperbilirubinemia occurs (Herring and Simpson). Simultaneously bile thrombi are found in the hepatic canaliculi as the tissues become bile stained.

Changes due to jaundice are most marked in the liver. It is firm, dark green and frequently nodular (biliary cirrhosis). There may be an increase of fibrous connective tissue. Intrahepatic ducts are enlarged. On microscopy in the central zone there may be large numbers of small bile ducts which are proliferating haphazardly and frequently surrounded by fibrous connective tissue. In these areas it is frequent to find periportal small cell infiltration. Splenic enlargement may be present, splenic vein thrombosis may occur. Portal blood pressure may be increased.

Microscopically changes in the liver due to jaundice are as follows: (a) bile pigment is deposited in the hepatic parenchyma, (b) hepatic cells show local necrosis, (c) bile canaliculi proliferate, (d) the hepatic parenchyma is infiltrated by small round cells, (e) periductal tissues demonstrate hyperplasia and fibrosis (Cantarow).

Despite organic changes due to obstruction to bile flow, hepatic cellular function continues to some extent (Mann and Bollman). However, a progressive unrelieved obstruction is usually fatal by reason of hepatic or hepatorenal insufficiency (Himsworth).

In an occasional patient when the bile ducts are obstructed the liver fails to form bile. Such a situation is not usually recognized clinically or pathologically. The existence, however, of "white bile" in the common bile duct is well recognized surgically (Aronsohn, Cantarow, Riegel, Ravdin et al.).

The presence of white bile or hydrops in the gallbladder is seen frequently when the cystic duct is obstructed. It has no bearing on the general prog

nosis However, the presence of white bile in the bile ducts is usually of grave prognosis (Bockus Lichtman Sodeman Weiss)

The etiology and pathology of this syndrome in which bile is not produced are obscure (Bernhard, Danner Paterni) It is known that it appears in longstanding cases of obstructive jaundice and usually, in patients with malignancy This has been reported in several cases of longstanding calculous disease of the common bile duct (Reigel Ravdin et al)

Experimental evidence indicates that acholia exists when obstruction to the bile passages co-exists with infection (Bernhard Danner Paterni) Infection undoubtedly accelerates the cycles in bile pigment change from pigmented bilirubin through nearly colorless biliverdin (Sickey) Experimental evidence also has shown that the bile duct epithelium both absorbs and dilutes the contained bile so that the residual fluid (except for calcium) becomes nearly isotonic for blood serum Hence the bile in the ducts is colorless

It is fair to assume also that the surface epithelial area of the biliary ducts is able to absorb and to excrete to deplete and to dilute Should bile no longer be formed, then, of course it can no longer reflux into the inter lobular spaces Therefore there may be no change in the icterus index Indeed it is conceivable that the blood bilirubin level could decrease Such observation may fallaciously influence the prognostic value of repeated determinations of the bilirubin levels Naturally bilirubin formation in other reticuloendothelial tissues must be considered in the clinical evaluation

The presence of longstanding obstruction opposing the normal excretory pressure of the liver may not depress the hepatic function of bile formation when the biliary passages can expand and simultaneously absorb sufficient fluid to keep biliary tract pressure below the secretory pressure of the liver However should the walls of the ducts be thickened by edema or by fibrosis secondary to inflammation the intrabiliary tract pressure may rise so that hepatic function is suppressed

Liver failure occurs following simple complete biliary obstruction due to choledocholithiasis There is usually seen progressive impairment in hepatic function Obstruction to bile flow associated with absence of bile in the intestinal tract may be followed by continued increase in serum bilirubin When hepatic failure occurs serum bilirubin may not continue to increase there is a decrease in prothrombin content a progressive decrease in serum albumin with reversal of A G ratio an increase in serum alkaline phosphatase and a variable change in the ratio of cholesterol esterified fraction

Because of the danger of bleeding liver biopsy is not frequently recommended in patients with obstructive jaundice However, it is essential to

recognize the gross changes in the ducts and the liver secondary to infection of the biliary tree

It is wise to evaluate all patients with jaundice whether or not of long duration to determine that the absence of increasing jaundice might indicate impending or actual hepatic failure

Such patients should be treated (a) with antibiotics to overcome the infection (b) by surgical drainage of the bile passages and (c) by adequate protein and glucose intake to reinforce hepatic cellular function

Whether or not the bile is permitted to reach the duodenum is not as important as the release of obstruction It is known that the liver cell can return to normal function even when damaged by extrahepatic biliary obstruction (Edlund) Secondary operation can be done later to reconstruct a proper route for bile flow into the intestines after hepatic function has been restored to normal (Hawthorne and Sterling)

F Bile Peritonitis

Extravasation of bile from its normal channels into the free peritoneal cavity can be fatal It has been demonstrated experimentally (Horral and Carlson) that 5 cc per kg body weight of sterile whole gallbladder bile of a dog will cause the death of the animal within 24 hours when this bile is injected intraperitoneally Intravenous injection of ox bile is fatal The cause for the fatality has been thought due to impurities bile acids bile salts cholesterol or bile pigments However, none of these have been demonstrated as definitely toxic in all experiments Positive proof concerning which factor is concerned as the toxic factor in bile peritonitis is lacking at present It is enough that the effect of bile on hematologic chemical and bacterial factors can produce blood volume changes to the point of physiological imbalance

Leakage of bile is well tolerated as a single episode However continued leakage involving the peritoneal cavity or the mesenteric roots can be rapidly fatal Any single perforation of the common bile duct will empty the bile passages When empty there is a tendency for the walls of the duct to be protected by adjacent tissue In the absence of any mechanical or physiologic obstruction to normal bile flow an accidental trauma to the common bile duct is not ominous

Peritonitis can occur following cholecystectomy due to bile escaping from the cystic duct stump

There are other sources for bile extravasation following cholecystectomy Accessory ducts to the gallbladder may be in the liver bed An accessory cystic duct may arise from the right or left hepatic ducts In these cases drainage will cease soon if there is free flow of bile through the distal portion of the common bile duct

McLaughlin and others have demonstrated the desirability of controlled drainage when leakage continues Continued extravasation of bile post operatively may indicate obstruction in the ducts

Findings in bile peritonitis include peritoneal reaction with cellular infiltration edema and necrosis Fat necrosis is not present The pathological features of bile peritonitis (or bile ascites) and pancreatitis are distinct In the first case fluid is bile stained and usually thin In the other there is no or little bile staining to the fluid and the exudate of the peritoneum is shaggy and dirty Bile peritonitis may show positive bacterial culture That of pancreatitis however very rarely shows positive culture Bile colored ascitic fluid is found in patients with longstanding obstructive jaundice In this state generalized icterus accompanies the ascites This should be differentiated from both bile peritonitis and pancreatitis

Bile drainage follows the removal of an indwelling T tube or catheter following surgery on the gallbladder or ducts In these cases a tract has been established by the drainage tube from the bile passages to the external surface The protective body mechanisms of fibrosis permit the formation of a fibrous and epithelial tract There is in addition a rapid growth of epithelium from the duct or gallbladder to surround the point whence the tube emerged Pressure of surrounding tissues usually obliterates the tract In the duct which permits normal flow into the duodenum it is unusual for drainage tracts to persist for more than 48 hours In fact most of these tracts seal within an hour of the removal of the tube

Leakage of bile from an accessory duct or other source may infiltrate between the peritoneal tissues of the gallbladder bed in the liver to form a pseudocholedochus cyst Subsequent escape or loss of bile from the cyst may inaugurate a rapidly fatal bile peritonitis Leakage often occurs into the subhepatic and subdiaphragmatic spaces The course however, of this localized process usually is to form a subhepatic cyst Because of enzyme activity and if secretory pressure of the bile continues it is feasible that spontaneous *perforation may extend through the diaphragm and the pleural* space Those cases reported of broncho-pleural hepatic fistulae however are usually of traumatic origin (Hewlett) It is true that in the presence of normal bile flow through the ducts these inflammatory and traumatic fistulae will usually be spontaneously obliterated

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5

LESIONS OF THE PANCREAS

PART ONE PANCREATITIS

A Pathogenesis and Diagnosis

Pancreatitis is an inflammation but not necessarily an infection Sixty per cent of the patients are younger than 50 years Sixty per cent are female

The (admission) diagnosis of acute pancreatitis is made correctly in only half the cases Clinical error occurs by reason of 1) failure to determine the serum amylase level at the time of admission, 2) tendency on the part of the physician to discount certain symptoms (Siler and Wulsin) and 3) the fact that pancreatitis may mimic a variety of other diseases

Clinical manifestations of pancreatitis are manifold Although the anamnesis is helpful, it may also be confusing For example in the treatment of abdominal pain the administration of morphine in certain individuals can cause pain and raise the level of the serum amylase, *per se*, without any actual pancreatitis being present (Wapshaw)

It should be kept in mind that pre existing coincident and complicating disease may be present and that neither pancreatitis nor the human protoplasm is static For example, Horsters found that patients could have an increased serum amylase due to duodenal ulcer pyloric ulcer, posterior wall gastric ulcer, carcinoma of the stomach, cirrhosis of the liver, tumor of the liver, parenchymatous hepatitis (or cholangiolitis), cholelithiasis cholecystitis obstructive jaundice and acute or subacute gastroenteritis In addition, patients with pancreatic disease are often found who do not have increased levels of amylase in urine or in serum (Morse and Achs)

Causes for pancreatitis as recorded in medical literature include

1 Infection through the lymphatics from distant focus
2 Infection through the blood stream from distant focus
3 Infection by direct spread of extra organic infection
4 Activation of bacteria in the normal pancreas
5 Extension of infection along pancreatic ducts from duodenum
6 Extension of infection from the bile ducts
7 Extension of infection from adjacent organ
8 Inflammation secondary to lymphatic or cellular extension of infection

9 Infection through portal system to liver and thence to pancreas
10 Associated with parotitis and infectious thrombophlebitis
11 Secondary to tonsillitis
12 Local (mechanical) stasis
13 Chemical activation of usually inactive ferments (from the autolysis of bile duodenal contents or pancreatic duct content)
14 Toxic products from general inflammatory or degenerative disease
15 Obstruction to (ligation) the pancreatic duct at the height of gastric digestion
16 Trauma (crush) to the pancreas (usually with blood vessel occlusion)
17 Injection of bile into the ducts
18 Injection of bile into pancreatic acini
19 Infected bile in the pancreatic duct or gland
20 Sodium taurocholate injected into the duct
21 As an unexplained sequel to focal liver necrosis
22 Regurgitation between the pancreatic and bile ducts associated with these factors (a) change in bile composition, (b) undue resistance such as from stone or spasm (c) inflammation, (d) mucus, (e) parasites (f) venous congestion as in cardiac decompensation
23 Excess dilute hydrochloric acid in the duodenum
24 Gastroduodenitis
25 Dietary indiscretions including alcohol
26 Duodenal contents in the pancreatic ducts in the absence of infection but in the presence of interductal regurgitation
27 Acute (or chronic) dilatation of the terminal portion of the duct in the presence of relaxed or dilated sphincter of Oddi
28 Duodenal obstruction associated with gastro-enterostomy
29 Duodenal stasis
30 Damaged valves in the papilla of Vater
31 Activation of trypsin in the pancreatic duct by regurgitated duodenal content
32 Diphtheria toxin
33 Extremely acid gastric juice
34 Alkalies
35 Calcium chloride
36 Formaldehyde or its products
37 White blood cells with bacteria (non viable) in the pancreatic duct
38 Hyaline necrosis of the pancreatic arteries and veins with metaplasia of pancreatic duct epithelium in the presence of free trypsin associated with local hemorrhage or thrombosis
39 Inspissation of the pancreatic secretion
40 Pancreatic tumor

41 Vascular degeneration or sclerosis in the pancreatic blood supply
42 Systemic toxemia
43 Local trauma (a) penetrating wound, (b) blunt force injury
44 Generalized trauma with reflex stimuli
45 Arterial occlusion secondary to arteriosclerosis or periarteritis nodosa
46 Excessive enzyme production secondary to extremely heavy meal
47 Prolonged (daily) administration of mecholyl
48 Acute or chronic alcoholism
49 Choledochoduodenal dyskinesia
50 Congenital anomalies such as diverticulum of the duodenum annular pancreas or pancreatic heteropia
51 Following prolonged administration of ethionine in rats (Brinkman and Rosenfeld) This is usually inhibited by the use of methionine (Popper)
52 Anaphylactic shock (or shock like state) produced by foreign proteins or histamine like substances (Franchini)
53 'Schwartzman' phenomenon occurring within the pancreas
54 Surgical trauma following dissection at the time of gastroduodenal surgery (Jones and Smith)
55 Normal or abnormal interductal reflux
56 Vagotomia
57 Mumps
58 Mesenteric venous (or arterial) thrombosis (local or massive)
59 Associated with penetrating duodenal ulcer (Bockus)
60 Drugs (morphine)
61 Coronary artery occlusion
62 Typhoid fever
63 Scarlet fever
64 Starvation postoperatively
65 Ligation of pancreatic duct
66 Any combination of minimal stimuli

Sixty six causes are too many It should be possible to assign proportionate value to (a) infectious (b) chemical (c) enzymatic and (d) obstructive components

There is co involvement of the hepato biliary system with certain types of pancreatitis This is amenable to therapy directed to the bile ducts and gallbladder

There are other (suppurative cystic fibrotic and neoplastic) types of pancreatic disease which are not due to disease of the biliary tract These are not benefited by therapy directed to the bile ducts and gallbladder

Infection may be the primary cause for pancreatitis by invasion through

the blood stream or by extension from the gallbladder through the hepato duodenal ligament Inflammation may also extend from a duodenal ulcer or from the common bile duct to the adjacent pancreas Infection of the pancreatic ducts in the absence of obstruction is rare

When there is interference to flow of bile the same may exist to flow of pancreatic juice and *vice versa* The mechanisms for flow of bile and of pancreatic juice are closely related anatomically and physiologically

Obstruction to the pancreatic duct and its blood supply in the presence of a secreting pancreas will cause edema of the pancreas with a rise in serum amylase This is most important and may be the *sine qua non* in the genesis of most cases of pancreatitis

It has been stated that reflux of bile into the pancreatic duct initiates pancreatitis due to irritation proportional to the bile salts concentration However the pancreatic duct does not hold more than 5 cc In most experiments relatively large volumes (10 cc or more) of bile are injected The injection of trypsin and almost any other substances in large volume and at great pressure will also cause pancreatitis

In order to avoid abnormal pressures rupturing the pancreatic duct Mann and Giordano Bisgard Baker and Anderson Wangensteen and co workers and Terjerina Fotheringham operated on goats, rats, dogs and cats so that bile reflux into the pancreatic duct was not under great pressure In most cases the passage of bile into the pancreatic duct under such conditions did not cause pancreatitis

There is no evidence that the mere presence of bile in the pancreatic ducts in the absence of pancreatic duct rupture or obstruction, can produce a rise in serum amylase or cause pancreatitis There is good reason to believe that if bile along with pancreatic juice should enter the interstitial tissues pancreatitis could ensue When Rich and Duff injected bile into the pancreatic duct (pressure of the injection was not recorded) tissue damage followed but there was no fat or vascular necrosis *In other words neither the existence of a common channel for bile and pancreatic ducts nor the pressence of interductal reflux are major etiologic factors in pancreatitis*

Finally acute pancreatitis occurs in patients in whom the pancreatic duct opens separately from the common bile duct (Popper Dardinski and Rich and Duff report two cases each and Johnstone New and Simkins one case each Sterling Mann and Giordano state that they have seen such cases) Reflux of bile is not necessary for pancreatitis In such patients however obstruction to pancreatic outflow may be present

The pancreas as any inflamed tissue may show edema or necrosis and polymorphonuclear leukocyte lymphocyte or phagocyte infiltration depending upon the stage duration and severity Fat necrosis appears as a

TABLE 8 DIFFERENTIAL DIAGNOSIS OF PANCREATITIS

	Acute Disease Edematous Chemical Enzymatic and Obstructive	Chronic Disease Suppurative Necrotic, Cystic Fibrotic and Neoplastic
Onset	Sudden	Gradual
Fever	Low grade or absent	Present may spike
Pulse	Normal	Rapid, weak
Blood pressure	Not changed	Frequently low
Pallor	No	Yes
Flushed	Occasional	Often
Cyanosis	Occasional	Frequent
Jaundice	Unusual	Frequent
Nausea and vomiting	Often	Recurrent and severe
Anorexia	Yes	Yes
Abdominal rigidity	Localized	None or generalized
Abdominal mass	No	Frequent
Palpable gallbladder	Only if acute cholecystitis present	Frequent in neoplasm
Abdominal tenderness	Yes	Not usual
Costovertebral angle tenderness	Occasional	Frequent
Cullen's sign, umbilical turgor	Occasional	No
Serum amylase Somogyi units	500-1500	200-500
Blood sugar	Normal	May be low or high
Calcium	Normal	Low

result of extrapancreatic and extra intestinal action of the pancreatic enzyme Hyperemia edema fibrosis suppuration, necrosis or cyst formation may be present in different areas of the pancreas

Recent mild or transient simple type of pancreatitis (edematous chemical enzymatic or obstructive) presents a clinical picture which differs from chronic disease of the pancreas (suppurative cystic fibrotic or neoplastic) Hemorrhagic pancreatitis may fit into either category (table 8) often being very severe

Complications frequently follow pancreatitis 1) diabetes which tends to be severe 2) retroperitoneal cellulitis, 3) abscess of lesser peritoneal space 4) generalized peritonitis, 5) obstruction to the colon, 6) infarction of the transverse colon and 7) gastric neurosis or gastritis

Distention of the lesser peritoneal cavity (fluid or necrotic material) may displace the stomach and duodenum, in producing gastric obstruction or peptic ulceration Gastrointestinal bleeding can occur (15 per cent) Coincident peptic ulcer is found in 5 per cent

Pancreatitis tends to recur (60 per cent) About 20 per cent of patients suffer a recurrent attack of acute pancreatitis within three weeks of the initial episode

In certain patients with pancreatitis without biliary tract disease, and in whom irreversible changes do not develop there can be complete recovery after the second or third attack In other types of pancreatitis recurrence appears with increasing physical deterioration

Operative mortality of acute (including recurrent pancreatitis) is from 12 to 16 per cent However operative mortality in patients with hemorrhagic necrosis of the pancreas is 60 per cent It is a very serious disease In the suppurative and cystic group, in whom operations were limited to drainage of fluid collections operative mortality is only 18 per cent

The presence of 'fat necrosis at operation indicates serious although not necessarily fatal prognosis Calcification or saponification of the plaque (representing enzymatic digestion) may begin within 24 hours Fibrosis usually occurs within seven days Microscopic examination of the areas suspected to be sites of fat necrosis is indicated, since gross appearance is deceptive In about half of the cases there is an excess of peritoneal fluid This fluid is clear yellow with interstitial lesions of the pancreas It is sanguineous when hemorrhagic necrosis of the pancreas is present In cases of interstitial hemorrhagic or necrotic pancreatitis inflammation may spread to the transverse mesocolon the root of the mesentery, the gastrohepatic ligament and the retroperitoneal tissues *Endamoeba coli* are recovered from the peritoneal fluid in many patients

Diabetes mellitus may accompany pancreatitis in its acute and chronic forms Hyperglycemia and hypocalcemia are both indicative of extensive pancreatic gland necrosis and may each be sufficient to contribute towards mortality in an individual patient

Since diabetes is about nine times as common in patients with biliary tract disease and occurs more than 40 times more frequently in patients with pancreatitis some etiologic aggravating or complicating phenomenon may be expected At the present time conclusive evidence concerning the basis for interdependency of diabetes gallstones and pancreatitis is lacking The weight of evidence tends to indicate that diabetes is not entirely *due to the amount of pancreas which has been damaged, and tends to in*duct fatty infiltration and degeneration in the liver which are present as a result of inefficient enzyme activity and production

B General Therapy

Selection of therapy for patients with pancreatitis depends upon accuracy of diagnosis Not only does this apply to the individual case but it also applies to the evaluation (in statistical hindsight) of therapy applied in groups of cases

There may be doubt about a clinical diagnosis when it is made by exclusion Actually positive diagnosis sometimes may be made only at opera

tion or at autopsy. Because there are many cases of presumed pancreatitis (as well as others in which the diagnosis has not been made) a wealth of ponderous, cumbersome and authoritative therapeutic armamentaria has overwhelmed the clinician.

1 Reported Methods for Therapy

Each of the following have been reported as favorably affecting the course of pancreatitis:

1. Avoid oral feeding
2. Intravenous fluid including glucose (a) with insulin, (b) without insulin
3. Gastric lavage, (a) constant, (b) intermittent
4. Ephedrine
5. Ergotamine
6. Calcium and potassium
7. No morphine
8. Adequate demerol
9. Barbiturates parenterally
10. Etamon
11. Nitrites
12. Theophylline
13. Atropine and/or belladonna
14. Magnesium sulfate orally four times daily
15. High protein, low fat diet
16. Frequent small feedings
17. Bland soft residue diet
18. Liquids only by mouth
19. *Bile salts by mouth*
20. Magnesium oxide
21. No alcohol
22. Pancreatin *(pancreatic extract) parenterally*
23. Adequate morphine
24. Reversed Trendelenburg position
25. Radiotherapy
26. Banthine and Probanthine
27. Hexamethonium
28. Paravertebral block (Novocain) *from T-4 to T-10, (a) right, (b)* left or (c) bilateral
29. Paravertebral and prevertebral sympathetic block at T-12
30. Splanchnic block on the right
31. *Soy bean trypsin inhibitor*
32. Intravenous serum albumin

33 Antibiotics such as penicillin, Aureomycin, Terramycin

Surgical therapy is also available Many procedures have been reported as successful in relief of pancreatitis

1 Cholecystectomy
2 Choledochostomy
3 Cholecystectomy and choledochostomy
4 Sphincter dilatation transcholedochal
5 Sphincterotomy transduodenal
6 Choledochoduodenostomy
7 Section of the choledochal nerves
8 Dorsal sympathectomy on the right
9 Dorsal sympathectomy on the left
10 Vagus section
11 Total pancreatectomy
12 Subtotal gastrectomy
13 Pancreatic duct transplanted to the duodenum
14 Double ligation of duct of Wirsung
15 Pancreatic duct drainage (a) transpapillary (b) caudal pancreatic ductostomy (pancreatostomy)
16 Pancreatolithotomy
17 Pancreatic cystectomy
18 Drainage of lesser sac through the foramen of Winslow
19 Choledochojejunostomy using a Roux en Y procedure with a cholecystostomy
20 Partial pancreatoduodenectomy followed by left thoracic sympathectomy
21 Splanchnic denervation on the right
22 Dorsal sympathectomy and splanchnic denervation on the right
23 Splanchnic section bilateral
24 Unilateral (left) or bilateral celiac ganglionectomy
25 Transduodenal sphincteroplasty
26 Drain pancreas drain pancreatic capsule and drain gallbladder or common bile duct
27 Open and close the peritoneal cavity without drainage
28 Appendectomy
29 Drain retroperitoneal region through posterior approach
30 Delayed cholecystectomy but drain open cystic duct
31 And for the complications (a) drain abscess of the pancreas (b) drain calcified and necrotic areas and (c) drain only exudative peritonitis

Here are more than 60 therapeutic methods for relief of pancreatitis This is almost absurd

2 Rational Management

Not only is the diagnosis of pancreatitis difficult but it is even more difficult to differentiate the stages of pancreatitis Yet, treatment depends upon identifying the changes which occur in pancreatitis as due to bacterial toxic enzymatic congestive or other causes Successful treatment requires systemic and organic rest, symptomatic relief with individualization

Intestinal symptoms may be due to the same cause as produced the pancreatitis or may be due to biochemical sequelae of the disease process

It is often difficult to establish whether pancreatitis is due to pancreatic disease or is secondary to some other abnormality Only when the biliary tract function is known to be faulty will it be feasible to manage pancreatitis effectively on the basis of biliary tract disease Because the sphincter of Oddi is a route for both bile and pancreatic juices it is wise to limit stimulus to bile flow and coincidentally limit stimulus to the pancreas Under other conditions therapy directed to the biliary tract may be useless

When the patient with pancreatitis is given adequate supportive therapy he will usually improve Meddlesome therapy may maintain the illness Because so many episodes of pancreatitis are self limited, coincident relief may be attributed to the therapy at the time disease manifestations improved

In management of pancreatitis the following should be done

1 Relieve pain
2 Counteract toxicity or infection
3 Relieve intestinal tract symptoms
4 Maintain blood cells and blood volume at normal levels and maintain alimentation electrolytes and fluids in normal balance

Management is organized in accordance with the severity of each illness There are four such groups

1 Mild pancreatitis
2 Pancreatitis associated with primary hepatobiliary tract disease
3 Pancreatitis associated with gastrointestinal or other disease
4 Pancreatitis accompanied by shock and toxicity

Therapy is directed mainly towards those features which have been observed to be the causes for mortality

1 Pancreatic necrosis
2 General toxemia
3 Abnormalities in fluid, electrolyte and cellular balance

C. Relief of Pain

In the patient under local anesthesia the pancreas and the pancreatic capsule can be stimulated painlessly. Distension of the pancreatic or bile ducts, however, does produce pain. Stretching of the peritoneum, mesenteries and other irritating stimuli (such as fat necrosis) are painful. Therefore therapy of pain in pancreatitis should:

1. Overcome pancreatic duct obstruction.
2. Relieve peritoneal irritation.

In most cases adequate doses of narcotics are very important. In a few cases pain may be aggravated by morphine. In those cases n-allyl morphine (5 mg.) will be useful. Severe pain will not be totally relieved by an anti-spasmodic. Papaverine (gr. ii every 3 to 4 hours) or nitroglycerin (gr. 1/100 every 3 to 4 hours) or intravenous procaine (0.1 per cent in 500 cc. normal saline) may overcome a portion of the pain component. The same is true of paravertebral blocks (chapter 10).

(Bilateral paravertebral injections are aimed at the ganglionated chains from the sixth to the tenth thoracic level. This is done with the patient prone, using a 3 inch long number 21 or 22 gauge needle for the multiple injections. The needle is passed perpendicularly to touch the tip of the vertebral transverse process. It is redirected cephalad to pass over this bone and at the same time medially to contact the lateral aspect of the vertebra. Six to 8 cc. of 1 per cent procaine is deposited.)

There is no reason to deny the patient adequate sleep and sedation. In patients who have other disease in addition to the pancreatitis, relief of pain due to those other organic causes is required. For example, in the patient with cholelithiasis and pancreatitis, pain may be due to stones impacted at the cystic duct rather than to the concurrent pancreatitis. The patient may have gastroenteritis, gastritis, mesenteric lymphadenitis, colitis or neuritis in addition to the pancreatitis.

Persistence of pain beyond several days may indicate complicating phenomena or additional disease. Persisting toxemia indicates that severe necrosis or abscess may have occurred. Pain relief usually accompanies subsidence of the disease.

D. Treatment of Infection

Bacterial, viral, toxic, protozoal, mycotic or non-specific infection can involve the pancreas and adjacent tissues by contiguity or through lymphatic and vascular channels. In addition, necrosis may permit further bacterial proliferation, particularly by the anaerobes.

Antibiotics are of inestimable value. Some physicians use duodenal intubation in order to obtain bile and pancreatic fluid for culture. The bacteria

obtained thereby may coincidentally be the invading organism in the pancreatitis Blood culture is only rarely positive

A broad spectrum antibiotic is very helpful Either Aureomycin or Terramycin may be given intravenously (1500 to 3000 mg per 24 hour period) Terramycin, erythromycin and streptomycin may be given intramuscularly The antibiotic should be effective within 72 hours The combination of penicillin with streptomycin is often spectacularly helpful Chloramphenicol or Neomycin may be used If gastrointestinal symptoms are minimal, sulfasuxidine or sulfathalidine is given for 7 to 10 days Aureomycin and Terramycin can of course, be continued orally The effect of the antibiotic is seen in the relief of the patient's toxicity, the subsidence of fever, pulse and leukocytosis Gas gangrene and diphtheria antitoxin may be indicated Gamma globulin may be helpful Antibiotic and antitoxic therapies are indicated in all types of pancreatitis

F Alimentation and Electrolyte Balance

The third factor in therapy is that of alimentation The patient with pancreatitis is often unable to utilize proteins and carbohydrates even if given parenterally

The quantity of fluid given to a patient depends upon individual (total estimated and measured) fluid requirements (a) glucose in water or saline (b) Ringer s solution, (c) electrolytes including potassium or calcium and (d) protein hydrolysate or plasma albumen

Parenteral alimentation is necessary if the patient is nauseated or vomiting It should be continued in the presence of abdominal wall tenderness or edema of the costovertebral angles A patient with acute pancreatitis often is anorexic or becomes nauseated at the sight of food Gastrointestinal symptoms may be so severe as to simulate mechanical intestinal obstruction

The individual who will not or cannot retain fluids should have nasogastric tube suction After 24 hours suction may be intermittent It is unusual for a patient with pancreatitis to require duodenal or gastric intubation for more than 48 hours The unusual case is one wherein severe or recurrent attacks have produced marked peritoneal edema or exudate

When improved the patient voluntarily requests food At first small amounts of cracked ice clear liquids or hot tea (one dram every 15 to 30 minutes) is given This is increased gradually After 12 to 24 hours the individual may take up to two or three ounces of clear liquids per hour After 24 to 48 hours diluted fruit juices are given Patients vacillate their desires among fruit juices milk tea and occasionally toast crackers potatoes jelly gelatin or custards The individual may progress then to a Muelengracht a bland or a low fat diet

Bowel obstruction can occur This is often seen in patients who have had previous abdominal operations Plastic exudate (resulting from pancreatitis) can involve an hernia sac to produce intestinal obstruction Serial survey x ray films of the abdomen may be taken to observe the degree of distention and the presence of abnormal fluid collections In those patients who have primary pancreatitis the return to normal alimentation is rapid and very few dietary restrictions are necessary In those patients who have associated or primary biliary tract disease it is wise to avoid fats chocolate cabbage and spices In those patients who have intestinal disease in addition to the pancreatitis shelled skinned foods and seasonings should be avoided Where there is associated hepatic disease or cardiopulmonary disease a high protein diet as soon as possible should be employed The use of lipotropic agents including choline methionine or lipocaic is frequently valuable

The patient with pancreatitis frequently has evidence of dehydration acidosis glycosuria hypocalcemia and jaundice Management of fluid balance is individualized Frequent measurements of electrolytes identify deficits due to excretion distention and abnormal loss The patient with hepatic and pancreatic dysfunction may demonstrate marked inability to handle glycogen In these cases small quantities of insulin may be indicated Sudden decrease in serum calcium may follow the saponification in areas of fat necrosis as the calcium is immobilized locally

Support of hepatic function by hypertonic glucose is not difficult in the acute case However in the patient with pre existing renal disease or with associated hepatorenal inadequacy additional high protein feedings such as are beneficial to the damaged liver can depress renal function Parenteral protein may not be properly utilized and frequently severe fatal uremia may be precipitated

F Blood and Cell Volumes

In the medical management of pancreatitis a most important facet is maintenance of the blood cells and volume Patients with acute pancreatitis have a tendency to dehydration and shock due to the local effect of the enzymes and to the persistence of the pain Enzymemia and enzymuria also alter blood cell concentration and blood volume Toxemia and shock are often reflected as changes in blood and tissue fluid volumes

It is awkward and frequently impossible to measure blood volume changes It should be done in any critical or problematical case Some information can be obtained from repeated cell counts hemoglobin and hematocrit determinations Careful clinical evaluation is important skin and tongue dehydration tissue turgor tachycardia blood pressure variations dyspnea and weakness are suggestive of a deficit in blood volume

Loss of circulating blood volume is a common cause for complications of pancreatitis not primarily due to the basic disease. Replacement by 250 cc to 500 cc (daily) of whole blood

1. *Will* reinforce cellular nutrition and compensate for fluid loss into inflammed areas,
2. Will *overcome depression of cell production* in damaged hemopoietic tissues,
3. Will maintain renal function,
4. Can improve cardiac output (which dwindling by reason of decreased blood volume may have initiated pulmonary edema)

It is not uncommon for patients with chronic (or recurrent) pancreatitis to have peripheral thromboses in the extremities or the mesenteries. This may be caused by damage to the splenic vein or it may be due to pancreatic enzyme (trypsin) dysfunction or an antitrypsin factor which inaugurates "sludge" phenomena. The extremities should be supported by elastic bandages in the presence of venous thromboses. If the prothrombin time *is normal, anticoagulants may be used*

G. Summary of Medical Management

Half of the patients with pancreatitis will recover within seven days of *the onset of the illness. Approximately 25 per cent of patients will take two* to four weeks before their symptoms have subsided. Of the remaining 25 per cent, half may die and the rest may manifest a chronic form of pancreatitis. For 75 per cent or more patients, then, conservative management is indicated

Etiologic factors are evaluated individually in each case. The pancreatitis is determined to be primary or secondary to disease of the hepatobiliary or intestinal tracts. Management is *directed towards*

1. Relief of pain
2. *Overcoming toxicity and infection*
3. Relief of gastrointestinal tract irritation
4. Maintaining normal electrolyte level, fluid balance, blood cells and volume

H. Surgical Management (*Chapter 11*)

It is important to realize that pancreatitis should be managed individually. In 15 per cent of patients complicating factors are present and surgical intervention may be necessary. The patient with pancreatitis offers a parallel to the patient who has peptic ulcer because in both cases surgery is performed to relieve complications

Complicating phenomena which are amenable to surgical therapy in

clude abscess necrosis, severe pain and obstruction to the bile ducts or the bowel

In the acute phase, surgery is directed to drainage of abscesses, to relief of obstruction to bile flow and to decompression of the pancreas and the intestinal tract Surgery in the chronic stage of pancreatitis is intended to combat these and to overcome pain

Surgical maneuvers which drain the biliary tract include 1) cholecystostomy and/or choledochostomy and 2) sphincterotomy (transduodenal or transcholedochal) Other maneuvers intended to bypass the papilla of Vater include cholecystojejunostomy and choledochoenterostomy Cholecystectomy is not intended as therapy for pancreatitis except insofar as it may eliminate infection or calculous disease of the gallbladder

The pancreas may be drained through the foramen of Winslow, across the mesocolon above the gastrohepatic omentum, through the stomach or through the left costovertebral angle (left subdiaphragmatic space)

Transperitoneally, on the left, caudal pancreatostomy (Link) may be efficacious The pancreatic duct may be drained transduodenally through the papilla of Vater or transabdominally by intubation of the ducts in the body or tail of the pancreas In certain phases of obstruction to the pancreatic duct it is possible to join the small bowel to the pancreatic duct or cyst Marsupialization is often effective In those cases where neither drainage nor bypass is suitable partial pancreatoduodenectomy is done with restoration of gastrointestinal and biliary tract continuity with or without anastomosis of the residual pancreatic duct Pancreatic calculi may be removed Calcinosis can be relieved only by excision

After all local phenomena have been repaired if symptoms persist and provided there is relief of pain by preliminary nerve block it may be feasible to remove nerve trunk plexus or ganglia

PART TWO OTHER LESIONS OF THE PANCREAS

A Pancreatolithiasis

Clinical manifestations of calculi in the pancreatic ducts or of calcinosis of the pancreas are minimal These calcareous deposits of calcium phosphate and carbonates are usually found in the head and body of the gland There may be as many as 300 and they may be as large as 1 by 5 cm They may be oval or branched like coral wherein the calcification has extended through several communicating ducts They are rarely facetted

Diagnosis is usually made during the course of radiographic investigation of digestive complaints These symptoms mimic gastric and cholecystic disease Indigestion and colic can both be caused by pancreatic disease Frequently coincident chronic pancreatitis and rarely absolute pancreatic

insufficiency occur Glycosuria and diabetes occur more commonly than in the normal individual

At operation the pancreas feels firm, irregular and nodular Diagnosis of a pancreatic disease is not entirely reliable by palpation alone It is not usually possible to palpate individual calculi Exploratory incision after reflecting the duodenum or after separating the gastrocolic omentum may be made into the gland to expose the duct and remove calculi (chapter 14) Following pancreatolithotomy drainage procedures may be instituted Rarely it is elected to perform pancreatectomy because of the severity of the pain and magnitude of the malnutrition

B Benign Tumors and Cysts

Cysts and islet cell adenomas are occasionally observed in the pancreas In addition, fibroadenoma lipoma myxoma chondroma, hemangioma and hemangioendothelioma may be found These may be pedunculated or be within the pancreas Intraductal lesions have not been reported to be observed at operation These, however may be expected as the incidence of operative radiography increases

1 Cysts

Benign pancreatic cysts are not common One case was found per 8000 admissions at the Mayo Clinic The sex distribution is about equal The middle age group is usually affected One case in a 5 month old infant has been reported Trauma is an important inciting agent in about half of the pseudocysts" Pancreatitis precedes a cyst in about 10 per cent of cases and cholecystitis in about 35 per cent It is not usual for a pancreatic cyst to present pancreatitis or cholecystitis as onset symptoms

The retention cyst is a true cyst It is lined by epithelium It may be single or multiple and the size varies from a barely visible lesion to one which may contain 20 liters of fluid The epithelial lining undergoes pressure atrophy in the large cysts This type accounts for 11 to 25 per cent of the cysts

The proliferative cyst or (cystadenoma) is a true cyst lined by epithelium It may result from growth of adenomatous tissue or from degeneration phenomena with formation of a multilocular cyst The majority are in the tail of the gland The wall is of fibrous tissue Contents are mucoid or bloody Papillary projections are present in the lining Malignant change frequently occurs The benign cystadenoma makes up 10 to 15 per cent of pancreatic cysts

The pseudocyst is outside the parenchyma of the pancreas usually in the lesser sac This group includes cyst like collections of blood or necrotic

material There is no epithelial lining The pseudocyst makes up about 60 per cent of pancreatic cysts

Congenital cysts are relatively rare Included in this group are dermoids and teratoma

Parasitic cysts are not infrequent They usually occur in conjunction with parasitic disease elsewhere The hydatid cyst is a large and smooth surfaced It usually has multiple daughter cysts Scolices may be seen clinging to the lining of the unopened cyst Cysticercus cysts are due to the *Taenia solium* These are multiple small and widely dispersed throughout the pancreas

Of the symptoms and signs of pancreatic cyst dull and intermittent pain is the most prominent occurring in 85 to 90 per cent of cases Vague epigastric fullness mild dyspepsia occasional nausea and vomiting and weight loss may be present Jaundice may occur in from 15 to 20 per cent of cases A mass is noted by the patient in the epigastrium in over half of the cases *The mass is palpable above the umbilicus and usually to the left* It is tense non tender and may move very slightly with respiration Roentgenograms are valuable in diagnosis (chapter 8)

Once the diagnosis is established operation is indicated for several reasons

1 The cyst may rupture with 60 to 70 per cent mortality
2 Malignant change may develop in a cystadenoma
3 Symptoms continue to increase

2 Heterotopic Pancreas

Heterotopic pancreatic epithelium may be found in the stomach, small bowel gallbladder splenic capsule and at other areas in the abdominal cavity In collective reviews of the recorded cases (now exceeding 589) about 32 per cent are noted in the duodenum the great majority concentrated in the vicinity of Vater s papilla About half of these occur as discrete nodules in the submucosa Pathologic changes in the nodule have been found similar to those in the pancreas including acute and chronic inflammation necrosis ulceration hemorrhage adenoma and cancer Symptoms occur inconstantly although most recorded cases are incidental autopsy findings

3 Annular Pancreas

Annular pancreas has been reported as a cause of duodenal obstruction in cases varying in age from two days to 74 years It consists of a band of pancreatic tissue arising from the head of the pancreas and encircling the duodenum

Annular pancreas may exist without symptoms In the majority of the

surgical cases the symptoms were those of a high intestinal obstruction In most cases roentgen ray examination will identify obstruction to the second portion of the duodenum

Two surgical approaches have been used in the management of this condition The first is to relieve the duodenal obstruction by excision of the constricting band of pancreatic tissue In some instances this has been followed by pancreatic fistula

The second method of relieving the duodenal obstruction surgically has consisted of some type of by passing operation This has usually been either a gastrojejunostomy or a duodenojejunostomy This is the considered treatment of choice

4 Islet Cell Adenoma

The islet cell adenoma (nesidioblastoma) is usually single, small, well circumscribed lesion in the body or head of the pancreas Symptoms of weakness, fainting and hunger due to hypoglycemia are characteristic However, the 'Whipple Triad" may be so severe that patients are considered to be neuropsychiatric problems (a) The attacks come in the early morning before breakfast or after severe mental or physical effort (b) During the attacks the blood sugar levels are always below 50 mg per cent (c) Recovery follows the administration of sugar by mouth or vein

C Malignant Lesions (Cf Also p 100, and Chapter 13)

Pancreatic neoplasm in the head of the gland, at the papilla in the pancreatic duct or at areas of heterotopia may compress the common bile duct Jaundice, therefore, may be an early symptom About 10 per cent of patients with primary carcinoma of the pancreas have chronic cholecystitis and lithiasis In the diabetic, malignancy of the pancreas occurs about 1 5 times more commonly than in the general population

The head of the gland is most often involved (70 per cent) Origin may be multricentric Most often it is hard and almost gritty It is a columnar or cuboidal cell adenocarcinoma originating from duct epithelium and arranged in irregular duct like spaces

Occasionally, the tumor is soft, fleshy and mucoid Microscopically, this is acinar cell adenocarcinoma

Carcinoma of the body or tail of the pancreas may be a hard irregular mass It occasionally is a soft vascular mass or a diffuse infiltration of the entire gland Microscopically it is a columnar cellular adenocarcinoma arising from ducts rather than from acini There may be an irregular alveolar pattern Stroma is variable The tumors of acinar origin may consist of rounded or polyhedral cells with very little stroma, occasionally like a round cell sarcoma

Local spread of pancreatic tumor involves the posterior wall of stomach the duodenal jejunal flexure, the spleen and the common bile duct Penetration and ulceration is not common Peritoneal seeding is seen if the involvement is in the body and tail Lymphatic spread from the head is to the subpyloric nodes From the body and tail lymphatic spread is to pancreatosplenic nodes and thence to gastric hepatic celiac, mesenteric and periaortic nodes Mediastinal nodes and lung metastases are not usual Vascular spread is through the portal system

Portal hypertension and ascites may be present Abdominal pain is produced by nerve sheath extension along the celiac plexus

Clinically, the disease is rapidly progressive, averaging seven months from recognition of symptoms to death Weight loss is common Abdominal or back pain appears as the initial complaint in only about half of the cases The upper abdominal pain radiates to the back is dull, aching and progressive It is aggravated at night by bed rest and is relieved occasionally by sitting or standing The pain may occur in an ulcer rhythm (10 per cent) Jaundice appears in two thirds of cases of which 80 per cent are associated with pain The painless jaundice picture is not truly characteristic in carcinoma of the pancreas

Mental symptoms of anxiety depression and insomnia appear in an occasional case Multiple venous thromboses are common and may be migratory Fatigue anorexia nausea vomiting constipation and diarrhea occur fairly frequently

Physical examination seldom reveals a palpable pancreatic mass A palpable liver is found in about two thirds of cases and, in about half, a palpable gallbladder Ascites occurs in about 15 per cent

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6

CLINICAL MANIFESTATIONS OF COMMON BILE DUCT DISEASE

A General Clinical Picture

The so-called predilection for the "fair, fat fecund female of forty" to have biliary tract disease is not exclusive Its adoption may lead to error A majority of patients it is true who have biliary tract disease are more than 40 years of age However nearly a third of the patients are younger In fact there are as many between the ages of 26 and 30 with biliary tract disease as between the ages of 41 and 45

In the younger age group with cholecystitis fallacious initial diagnoses may be made Such are abdominal migraine appendicitis Meckel's diverticulitis mesenteric adenitis peptic (duodenal) ulcer hepatitis nephritis pyelitis hyperemesis gravidarum and functional abdominal pain

In the older age group the diagnosis of biliary tract disease may be easy A characteristic symptom complex offers no difficulty However angina like pain or curious discomforts referred to other areas may completely mask intrinsic duct disease until the development of jaundice In many cases the only manifestation the patient may have in whom the common duct is packed full of stones is a "weakness" due to progressive hepatic dysfunction

1 Prodromal Manifestations

Indigestion and flatulence are universal duly being ascribed to over indulgence an extra spice a new food an argument or an incubating upper respiratory tract infection Nausea or belching together with a feeling of giddiness or fainting are attributed to similar causes Such is the basic symptomatology of cholecystitis It may be thoroughly disguised as a transient phenomenon Or it has occurred previously without sequelae and is expected particularly at family gatherings—with or without alcoholic refreshment These symptoms may become significant only after abdominal "fullness" has progressed to painful colic and nausea or belching to vomiting

In view of the many associated factors none can be identified as being "primary" or prodromal Infection metabolic disorders dietary inadequacies and excesses pregnancies and corsets have all been accused as

causes for gallstones and for cholecystitis True, there are so-called familial tendencies but the environment and the dinner table are factors in family life Typhoid fever is frequently followed by cholelithiasis with calculi containing the bacillus Eberthella We are not now exposed to that contagion so frequently yet gallstones are being *manufactured merrily by myriads* Even ascetic vegetarians and non pregnant women or children who do not wear corsets all have biliary tract disease The manifestations of cholelithiasis *including common duct disease are so protean that it is unusual* to have 'asymptomatic cholelithiasis "

In another category are congenital anomalies of the biliary tract associated *with jaundice The problem in such cases is to differentiate hemolytic* causes from intra or extrahepatic obstruction, inspissated bile, duodenal atresia and infection Other disease or anomalies such as fibrocystic disease *of the pancreas, lung or cardiac disease may be present*

The biliary tract may be the site of secondary manifestations of disease A perforated ulcer or appendix and residua of peritonitis may involve the *hepatic pedicle Gonorrheal peritonitis was a frequent offender before the* use of modern antibiotics Infections in the tonsils nasal sinuses colon or kidney can involve the bile ducts through lymph or blood routes Meta*static neoplasms often are located in the second portion of the duodenum* and thence involve the biliary tract Fungus and parasitic infestations may form abscesses or cysts external to or within the biliary tract

2 Symptoms

Symptoms of common bile duct disease coincide with and are overlapped *by abnormalities in the gallbladder, pancreas, stomach and liver* Abdominal pain with specific localization and radiation is often diagnostic When pain is followed by increasing jaundice localization is implied in the *common bile duct* (*However, digestive disturbances, fever* or chills and apathy are not always diagnostic of common bile duct disease even in the presence of jaundice) The presence of hematemesis or melena together *with jaundice may* indicate disease at the papilla Acholic stools mahogany brown urine and icteric sclerae are common, constant and reliable evidences of disease in the common duct

Loss of electrolytes through fistulae, vomiting or diarrhea, together with decreased intake or inadequate digestion can affect liver and bile-duct function Portal or biliary cirrhosis may follow repeated operative procedures for repair of the common bile duct, wherein thick scar has involved the hepatic blood supply Cirrhosis may be due to duct obstruction or may be superimposed

Frequent complications are the rule rather than the exception in common bile duct disease Internal biliary fistula or gall stone ileus may be first in

dication In other cases persistent external biliary fistulae and residual choledocholithiasis may be more serious than the original chief complaint Pancreatitis pancreatic cysts and tumors pleuritis pneumonitis liver and subhepatic abscesses and splenic vein thrombosis are complications which may appear without previous indication that the common bile duct is the source

3 Palpation of the Liver (Fig 39)

Franklin Hanger has indicated the essence of its value as one good feel of the liver is worth any two liver function tests Leon Schiff has indicated that palpation of the liver is just as valuable in jaundice Examination of the liver mass by palpation and percussion gives an idea concerning its size texture contour and sensitivity

An enlarged liver occurs in hepatitis obstructive jaundice due to stone and tumor in fatty infiltration and inconsistently in patients with cirrhosis of the liver The largest liver is found in patients with fatty infiltration If hepatic enlargement is absent in a patient with obstructive jaundice it is unlikely that tumor is present

The soft firm liver edge may be normal A blunt soft edge is often associated with cardiac insufficiency or metastases which may be deep within the liver An irregular nodular liver edge may represent tumor nodule or cirrhosis However the cirrhotic liver is firm rather than soft Liver nodularity is often produced by tumor mass which has broken through the

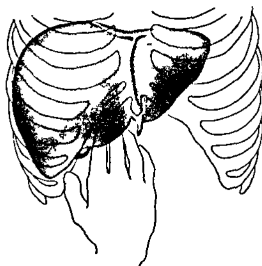


FIG 39 PALPATION OF THE LIVER (Courtesy of Chas Pfizer & Co Inc) One feel of the liver is worth two liver function tests —*Hanger*

capsule On occasion nodularity of the liver is caused by omental attachments which are residua of cholecystic disease (fig. 20a)

Tenderness is usually associated with hepatitis and is *often found with* abscess of the liver and subdiaphragmatic space. Localized *inflammation* may be represented by percussion tenderness

Information about the common bile duct may often be obtained by careful abdominal examination of the liver

B Primary Manifestations

1 Digestive Disturbances

Very prominent in the medical history of patients with abnormalities of the common duct are flatulence fullness and belching These are prominent also in all gastrointestinal disease *Differentiation from* esophageal ulcer disease or extra intestinal disease (such as pyelonephritis or renal calculi) cannot be made on the basis of the symptoms alone There is however a particular specific degree of pressure present beneath the right hypochondrium which may indict the biliary tract This appears more than one hour after completion of a meal and is associated with tenderness and often followed by a soreness Together with the fullness in the right abdomen is an increasing desire to move the bowels, to pass flatus and to "burp" This fullness is not relieved by body movements by heat or by removal of garments However time and various carminatives permit this to subside without residue

The patient may identify a particular item in the diet as the cause such are chocolate spices greasy foods pork lamb cabbage onion and salad dressing Frequently a patient notes that abstinence from these will eliminate the indigestion the fullness and repeated eructations associated with right subchondral pressure

The occurrence of actual or fancied constipation is a prominent symptom in many patients Epsom salts phospho soda saline laxatives often with calomel derivatives and bile salts compounds' can relieve their symptoms Enemas are not effective in relief of symptoms nor are such cathartics as cascara milk of magnesia castor oil or licorice powder The fullness pressure sensation is often followed by colicky pain Careful questioning is necessary to differentiate pressure from pain symptoms

2 Pain

It is evident from many reports of autopsy material and from clinical observations that a diseased gallbladder full of stones or a fibrotic pancreas with calcifications can be present without symptoms which equal the magnitude of the pathologic findings

The appearance of pain in disease of the biliary pancreatic system is due

primarily to distention of the ducts Fluid injected through drainage tubes left at operation produces the following types of pain

1 Distention of the gallbladder dull ache in right upper quadrant occasionally radiating to the right scapula

2 Distention of the pancreatic duct severe epigastric pain radiating to the back and sometimes to the left upper quadrant around the left costal margin to the left scapula

3 Distention of the common bile duct pain in midepigastrium radiating to the precordium or to the right scapular region

If the head of the pancreas is the seat of the chronic or acute inflammation pain may be periumbilical If the body or tail of the pancreas is involved pain may be in the precordium or may radiate from the left groin around to the left sacroiliac joint area

In some patients instead of pain a most remarkable reaction occurs to distention of the biliary pancreatic duct system Sudden profuse perspiration appears the face becomes suffused and the pulse is rapid and thready There is also a feeling of distention in the epigastrium or retrosternally (Doubilet et al)

Although pain in the right upper quadrant is commonly found with bile duct disease the biliary tract is not the only source for the pain Nor does absence of pain from the right upper quadrant exclude bile duct disease

Pain rhythms due to bile duct disease may simulate peptic ulcer even to relief with antacids Under such circumstances it is reflex pylorospasm which is being relieved Pain which awakens the patient during the night from a sound sleep is often due to bile duct distention This may be intermittent or constant localized to the xiphoid or to a small area below the right costal margin

Biliary duct pain is usually constant with periodic exacerbations over 1 to 3 minute intervals The pain can be piercing sharp and burning over several hours until the patient is in an agony of frenzy Girdling or constricting pain together with dyspnea is uncommon in biliary tract disease

Pain may be referred during exacerbations through the right upper quadrant and either to the epigastrium or around or through the right upper quadrant chest to the area between the lower border of the scapula and the ninth or eleventh dorsal vertebrae Anterior pain may radiate to the precordium Posterior pain may affect the shoulder neck or even the lumbosacral areas

When left abdominal pain is anterior it may be referred from the pancreatic capsule pancreas or ducts Pancreatic pain may be pseudo-agonal but with long intervals (hours) of freedom It is usually diffuse

Abdominal pain simulating gallbladder disease may appear in appendicitis hepatitis portal cirrhosis renal disease carcinoma abscess and pep

tic ulcer. In most cases, differentiation of appendiceal gastroduodenal and renal origins for symptoms may be made after a short period of observation and study.

Melancholy apathy, nausea and anorexia accompany a dull ache in the epigastrium or hypochondrium when hepatitis or cirrhosis is present. The pain of hepatitis is aggravated by movement or by eating, and there is accompanying tenderness. Tenderness and rigidity are increased and localized in the presence of diffuse or local infection.

3 Infection

Intra or extrahepatic obstruction to the duct system provides fertile field for infection. Bacteria are present in the gallbladder and appear in bile. In some cases infection may be introduced by surgery and through drainage tubes. Liver abscess cholangitis or choledochitis will occur more readily when bile flow is blocked. Under such conditions duct obstruction impedes drainage of infected material. Absorption then occurs to produce toxemia or bacteremia. Shaking chills occur. This is recognized as "Charcot's fever." It is often accompanied by intermittent and fluctuant jaundice.

Biliary tract infection is caused by bacterial flora of the bowel including *Streptococcus fecalis, Bacillus coli* and *Aerobacter aerogenes*. The anerobic Welch bacillus group are frequent secondary invaders and may be responsible for the fever and toxemia present in hepatic coma." Differentiation between duct infection liver abscess and pylephlebitis is difficult. Leukocytosis is common to all. In most cases antibacterial therapy by sulfonamides penicillin or the various "mycins" is ineffective until adequate surgical drainage has been provided.

Fever is present in 70 to 80 per cent of patients with acute cholecystitis and in only about 10 per cent of patients with chronic cholecystitis. Stones in the common duct are found in only about 5 per cent of patients with acute cholecystitis, may be present in about 20 per cent of patients with chronic cholecystitis and are found in at least 70 per cent of those who have a history of jaundice. It is not always possible to correlate presence of chills and fever to the presence of stones (table 9).

4 Jaundice (Plate One)

There may be clinical difficulty in differentiating hepatocellular jaundice from extrabiliary obstructive jaundice. There is no problem except in omission in the identification of the hemolytic types of jaundice.

Obstruction to the bile duct eventually causes liver damage. The clinical features of the disease furnish the best clues. Laboratory findings confirm but do not establish the diagnosis.

Failure of bile to appear in the intestinal tract may be due to the ob-

TABLE 9 ANALYSIS OF ACUTE AND CHRONIC CHOLECYSTITIS

	Acute Cholecystitis 185 Cases	Chronic Cholecystitis 328 Cases	Total 513 Cases
Fever	138	30	168
Leukocytosis	158	53	211
Dyspepsia	*131*	*262*	*393*
Dull pain to colic	162	195	357
History of jaundice	16	85	101
History of jaundice with common duct stone	7	51	61
Cholecystectomy done	133	315	448
Cholecystostomy done	52	13	65
Stones in gallbladder	*155*	*236*	*391*
Stones in gallbladder and common duct	6	62	68
Location of stones not stated	10	29	39

struction to flow or the failure in bile formation In severe forms of hepatitis an 'obstructive' phase appears in 20 per cent of patients Suppression of bile formation is seen in benign (rare) and in malignant (frequent) disease which also obstructs the bile ducts

Bile duct obstruction is followed by an acholic stool an increase in brown color in skin and urine concurrently with an increase in intracholedochal pressure It is known that bile duct epithelium (as well as the gallbladder) will absorb and excrete dilute and deplete bile which does not leave the common bile duct This together with bacterial activity produces white bile This exists as hydrops in the gallbladder It is of no general prognostic value However in the common duct the presence of white bile is of serious prognosis because it indicates actual or impending hepatic failure

Daily clinical and laboratory examinations of fecal and urinary bile pigment and urobilinogen can be supplemented by selected liver function tests Jaundice due to calculi is usually intermittent and fluctuant However when the serum bilirubin is stabilized or decreasing bile should disappear from the urine bile should appear in the stool and fecal and urinary urobilinogen return to normal Should a decrease in serum bilirubin not be followed by these coordinated and expected changes it is possible that hepatic failure rather than improvement is imminent

Once the diagnosis of obstructive jaundice is made with certainty preparations for surgical intervention should be made After the shortest quickest and best possible parenteral (or enteral) preparation surgical drainage of the bile passages should be accomplished If permanent relief of the obstruction can be accomplished it should be done However whether or not bile is permitted to reach the intestinal tract is not as im

portant as the relief of obstruction The liver cell can return to normal after back pressure is released At a later elective date secondary operation may be done to reconstruct a proper route for bile flow

Jaundice due to bile duct disease may be abruptly recognized although of gradual onset Patients with calculous cholecystitis who develop jaundice will not usually be a diagnostic problem An occasional patient with a past history and previous x ray evidence of cholelithiasis may develop jaundice due to tumor Even in such cases the likelihood of hepatocellular (inflammatory) jaundice is small and hemolytic jaundice is very rare On the other hand laboratory findings in a patient with jaundice who has had pre-existing cholelithiasis may be bizarre Unless positive evidence of primary intrahepatic disease is found, obstructive jaundice should not be permitted to continue beyond one week

Intrahepatic or "hepatic" causes for obstructive jaundice can exist as follows

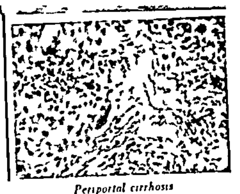
1 Acute and chronic cholangiolitis and interstitial hepatitis
2 Post—hepatitis bile stasis
3 Acute and chronic (viral) cholangitis
4 Schistosomiasis
5 Amebic hepatitis
6 Primary idiopathic scarring of the bile ducts as seen in periportal cirrhosis
7 Other causes toxic agents (hydrocarbons, alcohol), anoxia dietary deficiency debilitation secondary to systemic disease blood transfusion reaction and metastases

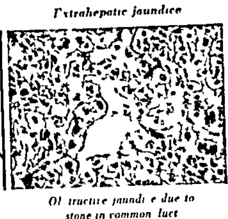
Under certain circumstances the clinical picture of extra hepatic obstructive jaundice is similar to that of cholangiolitic hepatitis (Watson and Hoffbauer) or other types of intrahepatic obstruction In addition laboratory findings may be similar Intrahepatic obstructive jaundice is seen in fewer than 1 per cent of patients under treatment with arsphenamine (Hanger and Gutman) thiouracil methyl testosterone and chlorpromazine (Lehman and Hanrahan)

The etiology of cholangiolitic hepatitis is not known Viral infections are suspected because of fever and constitutional prodromal symptoms which are 'grippal' in character The drug type of hepatitis is believed to be a hypersensitivity reaction It has been noted that jaundice may occur after relatively small amounts of a drug and that eosinophiles are prominent in periportal areas early in the course of the disease and may be increased in the peripheral blood smear In addition it has been reported that about 10 per cent of these patients may present urticaria or asthma particularly with chlorpromazine (Shay)

Microscopically liver biopsy (figs 38 and 91) in patients with cholangio-

turbed in 3 major areas as indicated with liver biopsies: *prehepatic jaundice* as with excess hemolysis of erythrocytes; *hepatic jaundice* as with hepatocellular derangement; and *extrahepatic jaundice* when there is obstruction of the biliary system.





Periportal cirrhosis

Extrahepatic jaundice

Obstructive jaundice due to stone in common duct

Reprinted from Pfizer Spectrum appearing in the Journal of the American Medical Association

litic hepatitis is characterized by the presence of inspissated bile in the smaller canaliculi Periportal regions have variable infiltration with mononuclear leukocytes and eosinophiles may be as prominent as in the drug induced disease Hepatic cellular damage may be present It is usually minimal The balloon type hepatic cell is frequently seen Hepatic cell necrosis is very rare but can occur Regeneration of cholangioles and of small bile canaliculi usually occurs in cholangiolitic hepatitis

It is not always possible to differentiate early in the disease between intra and extrahepatic jaundice However after several weeks the clinical course together with the laboratory and biopsy findings should permit accurate diagnosis

In intra hepatic obstructive jaundice the prodromal grippe' like manifestations may be minimal Clay colored stools dark urine and scleral icterus are most obvious Pruritus may be severe anorexia is common fever may be low grade but chills are rare Jaundice may be protracted, fluctuant and very intense even following such small total doses as 200 mg of chlorpromazine Clinical course tends toward recovery Fatality is more frequent following exploratory operation (Biopsy of the liver should be taken early in the operation to avoid confusion due to effects of anesthesia)

Except for the presence of jaundice physical examination may be normal in patients with intra hepatic obstruction Splenomegaly may occur in these patients it is unusual in calculous disease and obstructive neoplasia of the common duct Hepatomegaly occurs in all three states it constantly increases in patients with obstruction due to malignancy There is usually no pain and very little hepatic tenderness in patients with cholangiolitic hepatitis Cholecystography should be done preferably by intravenous method (fig 42)

In intra hepatic obstruction serum bilirubin may be high (30–40 mg per cent—prompt) Serum cholesterol may reach tremendous levels (800 mg per cent) The alkaline phosphatase is usually increased Recently it has been noted that cholangiolitic jaundice may be associated with a ratio between alkaline phosphatase (in Shinowara units) and total cholesterol (when greater than 300 mg per cent and divided by 100) which is less than 7 5 (Shay) Hyperbilirubinuria is present Urine and fecal urobilinogen may be absent Stools are acholic These may fluctuate If the obstructive phase is prominent serum flocculation tests may be normal If the hepatic cell is affected flocculation tests may be abnormal

Prothrombin time may be altered but will respond to therapy with vitamin K Total serum proteins in long standing cases are decreased mainly because of a subnormal albumin partition

Liver biopsy is particularly valuable This examination together with other observations of historical physical and laboratory examinations (par

ticularly in series), can establish the diagnosis of intra hepatic obstructive jaundice A high index of suspicion is necessary

Calculous disease of the biliary tract may be complicated by *cholangiolitic hepatitis* Diagnostic choledochotomy may be required

When the gallbladder is not functioning, onset of jaundice *due to calculous* obstruction may be very rapid It is often accompanied by pain and frequently by chills and fever, and is usually *intermittent All specimens of* feces and urine should be examined grossly Urobilin and bile pigment vary in intensity at irregular intervals between normal and acholic stools The presence of an occasionally clear urine during jaundice is suggestive of choledocholithiasis It is to be noted that *certain pigments of the porphyrin* cycle (Watson) may cause a dark urine and a positive test in the absence of bile

This type of intermittent jaundice is also seen in patients with primary (multifocal) *carcinoma of the liver In such cases an intraductal or intra* hepatic lesion may occlude certain segments of the liver while other segments *continue to function Sudden and increasing jaundice can appear in* the patient with a benign or malignant tumor of the papilla The gradual *character of the occlusion mechanism permits coincident increased intra* ductal pressure and coincident expansion of the gallbladder and ducts until *sudden decompensation occurs in liver cells*

Jaundice may be complicated by fever malaise rigors mental and physical *depression weakness apathy and marked anorexia Gradual onset of* jaundice is seen with hepatitis, cholangiolitis or lymphoma If inspissated *bile obstructs the bile ducts back pressure against the hepatic cell may be* increased to greater than 250 to 300 mm of bile pressure Liver cells can *not function under such pressure*

Abnormal cephalin cholesterol flocculation and other tests of hepatocellular *damage usually become apparent after a few weeks of complete* extrahepatic biliary tract obstruction These tests may be normal during *the first week or two of obstructive jaundice* Flocculation and similar tests *are positive early in the course of an acute hepatitis*

When choledocholitis or cholangitis is associated with choledocholithiasis as many as 30 per cent of patients will not show clinical jaundice In these *the clinical history of illness is from two weeks to two months or longer* Serum bilirubin may be high, normal or only slightly elevated Fecal and *urinary urobilogen may be absent*

Transient jaundice may appear after choledocholithotomy due to de*layed absorption of bile pigment*

Chronic disease of the duct is not common in patients who develop car*cinoma of the extrahepatic bile ducts* Jaundice is seen as the first sign in 7? per cent of these patients In these patients jaundice is followed by

dyspepsia abdominal pain and weight loss An enlarged tender liver is seen in about 25 per cent of these patients and in about half of these the gallbladder may be palpable About 10 per cent of patients with tumor in the extrahepatic ducts have co-existent cholelithiasis (chapter 4)

Except for the liver examination of the abdomen in a patient with jaundice is not always helpful in establishing a diagnosis Ascites a large gallbladder and a palpable mass may be present The mass may be soft and tender as may the enlarged gallbladder It may be nodular or firm localized or diffuse to merge with the contours of liver or other organ A large gallbladder in the absence of jaundice suggests the presence of cystic duct obstruction with hydrops or empyema of the gallbladder When the enlarged gallbladder is palpated in the presence of jaundice this suggests a normal gallbladder and the presence of obstruction due to malignancy in the distal common bile duct (Courvoisier specifically implicated carcinoma of the pancreas in his law ' concerning this) When the gallbladder can not be palpated in the presence of jaundice it indicates previous cholecystitis which has resulted in a shrunken gallbladder Choledocholithiasis is very frequent in this circumstance If the liver does not enlarge after two or three weeks of jaundice the cause for obstruction is probably inflammatory rather than neoplastic

Abdominal tenderness and rigidity may occur with cholecystitis, pancreatitis or primary ductal neoplasm Percussion auscultation may identify an anterior solid tumor Rectal examination may reveal pelvic metastases

Much information is obtained from the urine feces and skin Gross inspection can indicate whether bile is formed whether it is reaching the intestinal tract and whether it is being reabsorbed Full evaluation must be given to the effect of intestinal antiseptics which may decrease conversion of bile pigments by depressing activity of intestinal bacterial flora

To be carefully evaluated in the intact patient with jaundice are such lesions as lymphoma primary carcinoma of lung breast stomach and the skin (Addison s disease and carotinemia)

Jaundice in the patient who has had previous biliary tract surgery may be due to the surgery (ligation of the duct stricture from ligation of one wall of the duct) to sequelae (pseudo choledochus cyst cholangitis or residual calculi in the cystic duct stump migrating downward residual intrahepatic or choledochal calculi) or to a new process (tumor pancreatitis hepatitis or hemolysis) It is unusual for a patient with a choledochostomy to develop jaundice If this occurs in the presence of a functioning drainage tube a cholangiogram may indicate mechanical difficulties in position of the drainage tube Persistent jaundice may be associated with intrahepatic neoplasm or residual choledocholithiasis or due to hemolysis or hepatitis When bile drainage is not apparent through the lateral limb of the T

tube, mechanical obstruction can be due to (a) abnormal position with relation to the main hepatic ducts (b) inspissated debris in a very small tube, (c) kinking or (d) a defect such as a septum in the tube

Jaundice may be associated with hematemesis, black stools or massive gastrointestinal hemorrhage Anamnesis is of great importance Gastrointestinal hemorrhage which precedes the onset of jaundice may indicate a duodenal ulcer adjacent to the common bile duct Melena followed immediately by jaundice may be due to a fungating or ulcerating tumor at the papilla

Gastrointestinal hemorrhage may occur after jaundice has been present for a week or more In such cases hypoprothrombinemia, portal hypertension or gastroduodenitis may be present In portal cirrhosis, gastric hemorrhage from gastro esophageal varices may occur simultaneously with other evidence of hepatocellular decompensation In an isolated instance I saw gastrointestinal bleeding during a prolonged obstructive jaundice associated with splenic vein thrombosis and acute thrombocytopenia Another instance was in a patient who had seven bile duct reconstruction procedures over a four year period Five years after this he developed obstructive jaundice associated with fatal hematemesis

Obstructive jaundice is practically always associated with dilatation of the common bile duct A calculus is usually impacted at the distal termination of the extraduodenal common bile duct Therefore all of the duct between the site of impaction and the liver is dilated Stricture due to trauma is located where the common hepatic duct had joined the cystic duct Therefore although the extraduodenal common bile duct may be normal or small, the common hepatic duct between the stricture and the bile capillaries may be tremendously dilated

In those cases wherein cholangitis and/or choledochitis is followed by a long stricture the area of inflammation is thickened, stenosed and narrow while the duct between the stricture and the liver is only slightly dilated Dilatation of the bile ducts and capillaries in such cases is minimal Calculous disease results in a thick duct wall There is intramural and external fibrocellular reaction and edema As a result normal elasticity of the duct is impaired Accordingly the duct may be packed with stones yet not be dilated beyond 1 5 cm The trickle of bile between the calculi is as a rippling brook meandering across its *pebbled bed*

When obstruction is due to a malignant tumor or to accidental ligation the capacity of the bile duct may become tremendous and the common duct diameter may reach 3 cm

Obstructive jaundice is also seen as a sequel to pancreatitis, gastric resection, peptic ulcer perforation and exudate or adhesions in the *foramen of*

Winslow Under such conditions the common duct may histologically be normal

Obstructive jaundice and biliary colic also occur in children Obviously the index of suspicion is much less in the child of ten years of age than in the adult but the presence of obstructive jaundice at any age should be sufficient indication to exclude the presence of lithiasis and to relieve the obstruction Choledochostomy cholecystostomy and drainage of the liver ducts apply to the relief of biliary tract obstruction whether the child is six days or the adult 60 years of age There is an additional fact which should be emphasized A calculus in a duct does represent cause for obstruction It is important to determine that no additional disease exists A tumor may accompany a calculus several calculi may co-exist more than one segment of congenital atresia can be present

The common bile duct may be surrounded by pancreatitis and pancreatic calcification and the common hepatic duct may be compressed by lymphadenomegaly and exudate This may occur in actinomycosis or in nocardiosis In such cases the common bile duct is nearly obliterated and may not be identified because it is less than 2 or 3 mm in diameter Relief of this type of obstruction is obtained through adequate drainage of the necrotic material and by decompressing the liver by hepaticostomy

Drainage of liver and bile ducts can be accomplished through any dilated radicle even within the liver tissue If 30 per cent of the liver mass can be drained adequately many complicating phenomena of persistent prolonged jaundice such as hepatic coma or hepatorenal disease may be obviated A constantly draining bile fistula is not per se incompatible with life

5 Fistulae

a *Internal Fistula*

The internal bile fistula usually involves the gallbladder as cholecystocolic or cholecystoduodenal varieties and the common duct as choledocho-cholecystic or choledochal duodenal types The spontaneous internal bile fistula is usually due to calculi Occasionally malignant or benign gastric ulceration may involve the ducts or gallbladder It is not usual to recognize as such the clinical episode associated with the formation of an internal fistula Often the first indication that this exists is given on the appearance of the scout film of the abdomen The acute onset of intestinal obstruction due to a large gallstone may also be the first sign that an internal bile fistula exists

A cholecysto-enteric fistula usually follows empyema or hydrops of the gallbladder associated with localized mural gangrene and perforation Such fistulae involving the gallbladder tend to close spontaneously Symptoms

of peritoneal irritation are present in less than half of the patients However, the right upper quadrant is tender, often rigid Pain is localized and does not radiate Diaphragmatic irritation is frequent and the inflammatory process may activate a pleuritis or pneumonitis Reflex splinting of the right chest may initiate atelectasis In relatively few patients bile peritonitis may occur

Fistulae involving the common bile duct differ from those involving the gallbladder There is a large amount of tissue surrounding the common bile duct at its termination At the papilla, surgical probes and dilators readily deviate from the normal anatomical channel to establish a duct duodenal fistula These are usually unimportant because of the protection offered by adjacent tissues In some cases the surgical probe will perforate the pancreatic duct and pancreas Symptoms of pancreatitis may follow

Similarly, traumatic internal fistula may follow the impaction of a calculus at the distal end of the extraduodenal common duct This first forms a diverticulum then the fistula The diverticula formed under such circumstances have frequently been observed to cause pancreatitis Congenital fistulae between the pancreatic and common bile ducts have been reported in the papilla These are asymptomatic and of no known clinical significance

b External Fistula

Spontaneous perforation of the extraduodenal common duct may be minute and defy detection This closes rapidly after discharge of calculi Adjacent duodenum, gallbladder, hepatoduodenal ligament or stomach may seal the perforation Fistula formation between the common duct and *adjacent viscera is infrequent*

In most cases an external bile fistula is "lateral" rather than an "end" variety *An external bile fistula is very rare as a spontaneous event* Several cases have been reported of subcutaneous abscess formation representing perforation of the gallbladder which became biliary *fistula after incision* There is no known case of spontaneous external biliary fistulae originating from the common bile duct

External bile fistulae follow surgery involving the biliary passages In the great majority there has been drainage of the gallbladder or common bile duct In a few instances surgical procedures involving adjacent organs kidney colon duodenum or stomach may necessitate *the establishment of* a bile fistula

A fistula is established when the common bile duct is explored *(Only a* very few surgeons have advocated closure without indwelling drainage) The closure is not always absolutely bile tight so that a small amount of bile escapes around the drainage tube *This seepage* in addition to the presence of the tube inaugurates a foreign body reaction which forms a

tract Ordinarily when the drainage tube is removed from the normal postcholedochostomy duct, the tract leading from the duct will be occluded by the effect of pressure exerted from intra abdominal viscera Mucosa arising from the intestinal tract or gallbladder may line a chronic fistula This fistula can persist

Surgical choledochostomy is a lateral orifice It tends to close unless obstruction to distal flow is present Residual choledocholithiasis a papilloma at the papilla of Vater malignancy at the termination of the duct or stricture are the usual reasons a lateral fistula of the common duct would persist

Infection in a fistulous tract may form an abscess which requires drainage During such episodes purulent drainage may be accompanied by bile It is unusual for such a collection once evacuated to persist There are instances in which such a collection has formed a pseudocholecystic cyst Such can occur when the cystic duct lumen drains into the peritoneal pocket of the resected gallbladder The pseudocholecystic cyst may obstruct the bile duct

Bile drainage occasionally appears for a few days following gallbladder surgery This may come from the liver bed from an accessory hepatic duct at the cystic duct pedicle from the slipping of a ligature on the cystic stump duct or from injury to the common hepatic duct All types except the last will close spontaneously The last represents a technical error which has interfered with duct continuity and has partly obstructed the distal end of the common duct

Bile drainage may occur within 72 hours postoperatively following renal pyloroduodenal or hepatic flexure surgery During dissection of the duodenal stump and its subsequent closure a suture may occlude or penetrate the wall of the common bile duct A bile fistula following this type of surgery or the jaundice which may occur will not usually persist

Following pancreatoduodenal resection and following surgery to repair stricture of the common bile duct transient bile fistula almost always occurs A persistent bile fistula which occurs coincident with breakdown of the anastomosis is of secondary consideration to the presence of the intestinal fistula

Identification of bile in a fistula is usually easily made However bile is present in normal intestinal contents so that the fact of its presence may be inconclusive

6 Gastrointestinal Hemorrhage

Massive gastrointestinal hemorrhage usually does not originate within the biliary tract Reliable statistics concerning the appearance of melena in association with disease of the common duct are not available Certain

authorities (Bockus, Lusterman, Adler) indicate that about 25 per cent of patients with tumors of the pancreas, particularly those involving the papilla may show positive laboratory tests for blood in the feces.

It is not unusual to observe syncope or pain associated with a tarry stool which is followed after several days by jaundice. Typically this identifies a malignant tumor of the common bile duct which in ulcerating through the papilla has also occluded it. This order of symptoms is also seen following gastrectomy when the duodenal stump has been closed too close to the common bile duct and jaundice becomes apparent while a tarry stool continues due to the operative trauma.

Any ulcerating neoplasm eroding the duodenal wall and obstructing the common bile duct may *produce gastrointestinal bleeding and jaundice*.

Biliary tract bleeding usually appears in the stool. It is only rarely that *it appears in vomitus. When jaundice appears together with hematemesis* it is often not due to primary disease in the bile ducts. Hepatic cirrhosis may *be associated with icterus and simultaneous bleeding from esophageal* varices.

Another instance of massive hemorrhage associated with pre-existing jaundice occurred in a patient in whom a pseudocyst of the pancreas formed following pancreatitis. In this case, hemorrhage appeared through the "T" tube as well as in the gastrointestinal tract. The bleeding was recurrent over several weeks and finally was massive enough to be fatal. It was found that the action of the pancreatic enzymes in the pseudocyst had eroded the right hepatic artery into the right hepatic duct.

Congenital varicosities, hemangiomata and aneurysms within the common duct and adjacent to it have been reported as associated with gastrointestinal bleeding, with and without jaundice.

Another confusing picture is manifest in a patient with multiple liver metastases or primary liver malignancy. I recall a patient whom Dr. A. Katz hospitalized on suspicion of hepatitis. Occult blood was found in stools which were intermittently acholic. Jaundice increased and bleeding continued. Gastrointestinal x-ray study revealed *evidence of duodenal ulcer*. Cholecystography showed a functioning gallbladder which in this instance was among the 4 or 5 per cent error. The patient was bleeding from multiple and superficial ulcerations from the stomach, small bowel and the cecum. Jaundice was both intrahepatic and extrahepatic in origin and due to a *primary (multicentric type of) malignant hepatoma*. The gallbladder was shrunken around one medium sized calculus. In this patient the low *prothrombin time was responsible for the gastrointestinal bleeding*. The malignancy was responsible for the jaundice.

Jaundice may be accompanied with *gastrointestinal bleeding per se*, as a result of hypoprothrombinemia or because of increased portal blood

pressure It must be noted that the transfusion reaction associated with hyperbilirubinemia should not be confused with organic causes for jaundice

7 Neoplasm of the Biliary Tract

Most cases of primary malignancy of the liver occur in the fifth to seventh decades of life

Clinical signs and symptoms usually are not clear The most common presenting symptoms are weight loss abdominal pain and jaundice Gastrointestinal symptoms such as dyspepsia nausea and vomiting are present in 30 per cent of cases Physical examination reveals a palpable liver in 70 per cent a separate abdominal mass in 35 per cent and right upper quadrant tenderness in 25 per cent

The clinical diagnosis of hepatic cancer is possible The most important symptoms are right upper quadrant pain anorexia and weight loss The commonest physical findings are ascites jaundice and hepatomegaly There is occasional nodularity of the liver if ascites is absent The liver may be tender Spider angiomata splenomegaly and massive gastrointestinal hemorrhage are occasionally present Hepatic tests indicate both chronic and active hepatic cellular damage Positive cephalin flocculation and thymol turbidity reactions occur early Zinc sulfate flocculation and gamma globulin turbidity also appear at the time that there is a reversal of the albumin globulin ratio The alkaline phosphatase level is not of any differential value Blood cell counts show anemia in about 35 per cent of patients with occasional leukocytosis Urinalysis is normal

Diagnosis may be made by liver biopsy exploratory laparotomy or examination of sediment from ascitic fluid The patients die a relatively short time after symptoms appear

In cancer of the extrahepatic bile ducts the clinical picture is rapid and fulminating Jaundice is evident in 75 per cent dyspeptic symptoms in 50 per cent abdominal pain in 40 per cent and weight loss in 40 per cent Palpable liver is present in 50 per cent of patients a right upper quadrant mass in 50 per cent and right upper quadrant tenderness in 25 per cent

The gallbladder is not enlarged in patients with hepatic duct carcinoma Twenty per cent of tumors at the junction of cystic and hepatic duct 60 per cent of tumors in the common duct and 85 per cent of tumors at the papilla are associated with an enlarged gallbladder Chronic disease of the gallbladder is uncommon

The onset of duct carcinoma is that of progressive jaundice preceded by malaise and accompanied by weight loss Biliary colic occurs in 15 per cent pain under the right costal margin or in the epigastrium in 50 per cent This pain suggests calculous obstruction of the common bile duct so that the true nature of the lesion may not be suspected prior to laparotomy The

age of patients with bile duct carcinoma is somewhat higher than that of patients with carcinoma of the gallbladder Half of the patients die within the first two weeks after operation

Ascites or a palpable mass may be found with carcinoma of the pancreas but seldom appear with chronic pancreatitis Physical findings are scarcely helpful Upper abdominal tenderness or rigidity may occur with chronic pancreatitis also but are usually seen during an exacerbation of the disease and subside rapidly Jaundice is transient with the benign condition

Stimulation of the pancreas with secretin prostigmine and other drugs is likely to lead to elevation of serum amylase values in patients with pancreatitis or duct obstruction In carcinoma of the pancreas such stimulus produces no change

Ligation of the pancreatic duct in the experimental animal results in a rise in the serum amylase and lipase for a few days then a decline to low values Similar findings are present in carcinoma of the head of the pancreas with complete obstruction of the pancreatic duct The serum amylase and lipase are usually low when the patient is first seen Contrary to reports in the literature, elevated serum lipase is not often observed in cancer of the pancreas When the pancreatic duct is partially obstructed by cancer the serum amylase may rise to unusual levels This is especially true when the mesenteric vein is eroded by the tumor Under these conditions widespread liver metastases are usually found

In cancer of the head of the pancreas, the pancreatic duct is usually obstructed before jaundice occurs Pancreatic juice is then absent Obstruction to the supraduodenal common bile duct by carcinoma does not interfere with secretion of pancreatic juice

Routine laboratory data has proved valueless There are no consistent changes in the hematologic status in these patients

Abnormalities suggestive of malignancy (abnormal contour of bile passages) may be seen on x ray and cholecystogram (chapter 8) X rays interpreted as showing malignancy are seen in patients with far advanced carcinoma

Tumors of the duodenum are rare and difficult to diagnose Surgical treatment is employed in 15 per cent of benign and [illegible] per cent of malignant cases Pancreatoduodenectomy results in an operative mortality of [illegible] per cent

Carcinoma of the gallbladder is difficult to recognize and rare to cure It may be found in 0 4 per cent of all autopsies and 1 2 per cent of all patients with biliary tract disease Cholelithiasis is seen in from 30 to [illegible] per of cases of carcinoma of the gallbladder in post mortem studies (table 10) Other clinical investigations indicate that from the surgeon's viewpoint incidence of cholelithiasis is much greater in gallbladder carcinoma many reports indict up to 92 per cent of carcinoma with preceding cholecystitis

TABLE 10 CHOLELITHIASIS AND CARCINOMA OF THE GALLBLADDER

Author	Autopsies Total Number	Number of Cases of Carcinoma	Per Cent Carcinoma	Per Cent Cholelithiasis with Carcinoma
Kirshbaum and Kozoll	13 330	62	0 46	30 6
McLaughlin	9 523	101	0 073	38
Fieler	26 895	—	0 26	41

Pain dyspepsia weight loss and a palpable mass accompany jaundice in patients with carcinoma of the gallbladder Constant pain is present in the right upper quadrant and epigastrium It may be associated with belching and indigestion Anorexia and vomiting appear first intermittently and become very severe at about the time that jaundice occurs Weight loss is rapid over several weeks to months Jaundice may be sudden in onset persistent and usually associated with bleeding infections and rapid clinical deterioration due to hepatic metastases rather than common duct occlusion

Carcinoma of the gallbladder is frequently found unexpectedly during elective operation for cholelithiasis Careful examination of the resected gallbladder may reveal neoplasm Five year cures of gallbladder malignancy are uncommon

C Relation of Other Disease to Disease of the Common Bile Duct

It has been noted that digestive disturbances abdominal pain chills and fever may be associated with jaundice bile fistulae and gastrointestinal bleeding These indicate that disease has affected the biliary tract and particularly the common bile duct However disease of the bile ducts may exist for years without any of these primary symptoms The patient with residual choledocholithiasis may manifest only languid apathetic melancholy or may feel weak and complain of swollen ankles or of constant recurrent pleurisy or perhaps merely of an intermittent backache Symptoms of common bile duct disease can appear as a result of effects from the gastrointestinal tract the liver the cardiorespiratory apparatus the skeletal system and the urinary tract Similarly disease of the biliary tract may affect these systems The far reaching manifestations of common bile duct disease depend upon its chronicity for the acute episode is almost always recognized

1 Hepatorenal Disease

The origin of the term hepatorenal syndrome is lost in a mass of literature of the 1940 s concerning lower nephron nephrosis and renal insufficiency Many patients who had been exposed to burn blast and immersion injuries were found to have jaundice and anuria Excessive muscle and blood

destruction produced hepatic *necrosis* and tubular nephrosis. Prior to World War II when bilirubin appeared in the kidney *tubules* it was classified as "bile nephrosis." Subsequent to 1946 such deaths were classified as due to "hepatorenal syndrome."

Actually this syndrome is an all inclusive term covering such clinical and pathological states wherein liver and renal disease *may co-exist*, be interdependent or be mutually deleterious. It exists as: (a) *hepatic insufficiency* followed by renal shutdown, (b) renal insufficiency with secondary hepatic failure, (c) jaundice (obstructive or hepatocellular) followed by renal failure and (d) nephritis associated with hepatic coma.

a. Blood loss in cirrhosis associated with hemorrhage may establish sufficient hypotension to inaugurate lower nephron nephrosis.
b. Continued loss of albumin through the kidney may be followed by failure of the liver to *produce it*.
c. Excessive amounts of bilirubin together with other noxious catabolites formed during jaundice may be followed by a *chemical nephrosis and* a focal (toxic) nephritis.
d. Severe nephritis (and/or nephrosclerosis) in which there is loss of chlorides, potassium and total base together with additional failure to retain proper fluids can precipitate hepatic insufficiency by reason of hypovolemia alone.

Hepatic complications of biliary tract disease may be manifest as cholangitis, hepatitis, liver abscess and other inflammatory states. Cirrhosis of the obstructive biliary variety may provoke the more common portal cirrhosis. Hepatic failure may occur after long continued obstructive jaundice. Because of the secondary hepatocellular damage there may be a fallacious picture of primary hepatitis. Hepatic coma should not be the first sign observed by the clinician in a patient with common bile duct disease.

Renal tubular dysfunction is only rarely a manifestation of primary choledochal disease. Degenerative phenomena, infection and toxic manifestations may just as easily involve kidney as liver.

An unexpected and usually unforeseen interrelationship between biliary tract and the genito-urinary system is that of a technical nature. Procedures involving kidneys may involve the duodenum or pancreas and disturb biliary tract innervation. Technical procedures to the hepatobiliary tract may traumatize the kidney or perirenal tissues. These are rare and are seen after extensive resections for malignant disease involving either system.

a. Inflammatory States

Bacterial inflammation at the papilla such as cellulitis or carbuncle is rare. Mucosal and glandular infection accompany duodenitis. The papilla

becomes damaged otherwise only by trauma such as that due to calculi Acquired stenosis is traumatic in origin Symptoms accordingly are minimal except if obstruction to bile flow occurs

In the presence of inadequate bile flow together with infection of the bile passages cholangitis or choledochitis may be followed by cholangitis lenta cholangiolitis hepatitis and intra or perihepatic abscess Signs of suppuration including toxemia chills fevers and fatigability may be supplemented by abdominal tenderness pain and jaundice

These clinical episodes may closely mimic viral (infectious) hepatitis One phase may merge into the other and differentiation is difficult Viral hepatitis and bacterial cholangitis may co-exist and differentiation be made only by the surgeon or the pathologist However viral hepatitis should be differentiated from liver abscess and that from cholangiolitis choledochitis Laboratory data may not be helpful Differentiation is best made on the basis of history and physical findings (table 11)

Severe inflammation of the liver may mask pre existing and primary disease of the common bile duct Stricture of the common bile duct may permit sufficient bile flow have a minimal inflammatory reaction and not be associated with pertinent digestive disturbances Hence although the stricture may be of years duration its first significant symptoms may be failure of hepatic reserve and the appearance of jaundice fever or severe malaise

b Catarrhal and Septic Jaundice

Catarrhal jaundice the result of inflammation of the papilla of Vater with consequent ascending infection as postulated by Virchow is ex

TABLE 11 DIFFERENTIATION OF LIVER INFECTION

	Hepatitis	Liver Abscess	Cholangitis
Age of patient	Young	Middle age	Older
Character of fever	Plateau	Spiking	Intermittent
Chills rigors	Rare	Frequent	Occasional
Jaundice	Minimal	Unusual	Common
Physical depression	Marked	Minimal	Not unusual
Pre existing lithiasis	Uncommon	Uncommon	Common
Pre existing surgery	Uncommon	Common (except amebic)	Usual
Previous hepatitis	Common	Rare	Uncommon
Duration of illness	Weeks	Days	Months
Pain tenderness (RLQ)	Occurs	Common	Uncommon
Hepatomegaly	Common	Uncommon	Occurs
Bromsulfalein excretion	Decreased	Normal	Usually decreased
Flocculation tests	Positive	Normal	Variable

tremely rare The pathogenesis of cholangiolitic biliary cirrhosis is still controversial The process is considered by some as a descending cholangitis and by others a hematogenous infection which involves the bile canaliculi The pericinar or cholangiolitic form is characterized by prolonged, severe jaundice and an enlarged liver and spleen Symptoms are usually part of the manifestations of cholangitis complicated by focal hepatic change It is most *frequent in patients who have had biliary tract surgery* or disease for many years In its mild form there may be mucous debris and a pale orange color to bile draining from a chronic fistula Clinical differentiation is difficult The diagnosis is made by liver biopsy

Septic jaundice usually follows severe infection or surgery such as gastrectomy or appendectomy At necropsy the liver may show fatty degeneration with or without liver abscess or pylephlebitis It may be that only *minimal signs of hepatitis are found* Bacteriologic examinations indicate the *Staphylococcus aureus* the *Bacillus aerogenes* and the Welch bacillus

c *Liver Abscess*

Liver abscesses are amebic (60 per cent) or pyogenic (40 per cent), single or multiple Solitary abscess mortality is about 30 per cent Untreated multiple liver abscesses carry mortality between 90 and 95 per cent Open drainage will improve mortality rate to between 20 and 30 per cent

The portal vein is the most common single route for infection In 11 to 12 per cent the infection originates in an appendiceal abscess Rectal intestinal or splenic lesions may also be the site of origin In 13 per cent of cases liver abscess is derived through the hepatic artery from an osteomyelitis, furuncle or endocarditis Infection may spread to the liver by contiguity from cholecystitis subphrenic abscess perforated peptic ulcer leaking duodenal stump lung abscess or perirenal abscess

Trauma to the liver, of either the penetrating or non penetrating variety is responsible in 5 to 10 per cent of liver abscesses (Ochsner et al)

Signs and symptoms are often vague There are malaise and anorexia leading to weight loss and moderate anemia Dull aching pain may be in the right upper quadrant the epigastrium, the right scapula or the right shoulder Fever is a constant finding, it is remittent intermittent or continuous Chills are usually associated with *secondary infection* Leukocytosis is usually moderate (Table 11)

On physical examination tenderness is elicited over painful areas of the costal margin or liver by pressure and percussion *Signs of peritoneal irritation* may be present Respiratory movements may be decreased on the affected side Liver dullness may be increased Physical findings depend on the location of the abscess within the liver With an abscess high on the lateral surface of the right lobe there is tenderness on fist percussion or

pressure between the ribs If the abscess is in the center of the right lobe there is often persistent intense pain in the right loin With an anterior abscess there is tenderness below the anterior costal margin

Hepatomegaly is present in 70 to 85 per cent of the cases Jaundice is uncommon in solitary abscesses unless the bile ducts are obstructed Jaundice is most frequent (25 per cent) in association with multiple abscesses of pyelophlebitic origin Abnormal physical signs may be noted in the right lung field if the abscess involves the diaphragm

Blood culture is generally sterile Leukocytosis is present The organism most frequently recovered from the abscess cavities is *B coli* When multiple abscesses of the liver occur secondary to biliary tract disease obstruction is present in the bile ducts

d Subdiaphragmatic Abscess

As a result of maneuvers during bile duct surgery and postoperative seepage suprahepatic and infrahepatic spaces may be filled with exudate atelectasis and pleural effusion may occur on the right and a subdiaphragmatic collection exist Other causes for a subdiaphragmatic abscess are

1 Leakage of bile postoperatively
2 Biliary tract perforation
3 Pancreatitis
4 Appendiceal abscess
5 Peritonitis
6 Genito-urinary tract infections

Responsible organisms vary but most frequently these are the colon bacillus streptococci staphylococci and gonococci

Clinically the following are found in patients with subphrenic abscess

1 Localized tenderness (depends upon location of abscess)
2 Hiccough
3 Limitation in inspiration (without dyspnea tachypnea and orthopnea) Pain on inspiration may be referred to or be primary in the shoulder region
4 Increase in percussion dullness (difficult to elicit)
5 Fever usually septic in character (between 100 and 103)
6 Extreme fatigue weakness and moderate anorexia

Fluoroscopic limitation and deformity in movements of the diaphragm are significant Localization of an abscess by aspiration (to confirm the diagnosis) is not recommended because of the great danger in disseminating the infection

The right inferior subhepatic space usually contains blood bile and other exudate following biliary tract surgery The usual cholecystectomy has up to 250 cc of exudate in the right infrahepatic space This will be increased

if needle cholangiography be done. The operation is followed by a splinted right costal cage because of retraction maneuvers as well as the presence of abdominal pads against viscera during and abetted by the dissection. Immediately postoperatively, decreased respiratory excursions may interfere with normal diffusion through peritoneal surfaces. Accordingly lymphatics which penetrate the diaphragm into the pleural spaces become filled and pleural effusion often develops. *Involvement of the adjacent subdiaphragmatic spaces* depends upon the amount of soiling during and following surgical manipulation, the degree of tissue trauma and the frequency of bacterial infection.

Secondary infection may spread to 1) the left posterior subhepatic space through the foramen of Winslow, and 2) the right posterior suprahepatic space. Liver abscess of cholangiolitic origin may perforate into any subdiaphragmatic space.

e. Cirrhosis

Longstanding biliary tract obstruction, particularly that due to residual choledocholithiasis, is often followed by biliary cirrhosis. The cirrhotic state may develop in an interval of weeks to years. Due to the regenerative capacity of the liver lobule clinical recognition may be delayed until jaundice *or bleeding supravenes. Diagnosis of biliary cirrhosis may be made by liver* aspiration or at operation.

In patients who have had several operations, whether for calculi or strictures, there may be an extrahepatic portal circulation obstruction which is due to scar tissue in the hepatic pedicle. Portal hypertension due to disease at the liver hilum is manifest by ascites, hematemesis, melena and abnormal collateral circulation. Splenic vein thrombosis may occur. Simple cholecystectomy may be followed by splenomegaly, purpura and evidences of hepatic dysfunction. I have seen recovery follow conservative management. In a case reported by Strelinger splenectomy was necessary for the patient's recovery.

The course of a patient with cirrhosis is unpredictable. Patients can recover from severe symptoms. The multiplicity of hepatic cellular function plus the fact that there is mutual overlap within the cell may permit adequate hepatic function before the vital centers fail. Yet, depletion of hepatic glycogen alone may be fatal in some cases. In addition, total occlusion of hepatic arterial and portal venous supply is followed by focal hepatic necrosis. Partial occlusion of these vessels may not be fatal, particularly if antibiotics are given to overcome bacterial anaerobic invasion. However, Terramycin and Aureomycin increase protein anabolism and may increase fatty infiltration, both of which are harmful and may be fatal.

f Acholia

The absence of bile (acholia) from the intestinal tract is usually associated with obstruction to the bile passages. An additional cause is the failure of the liver to produce bile. This is often a premortem event.

The few reports in literature concerning acholia are vague. The etiology for and the pathology of this syndrome in which bile is not produced are obscure. It is known that it appears in longstanding cases of obstructive jaundice and usually in patients with malignancy. It has been reported in several cases of longstanding calculous disease of the common bile duct.

Of similar character is the "obstructive" phase present in some 20 per cent of patients with hepatitis. The acholia in these cases has not been satisfactorily explained. Exudate may block the periportal region at the junction of bile capillaries and duct or there may be a rupture of this canal simulating the "regurgitative reflux" mechanism. The best indicator regarding flow of bile and its production is in serial determinations of serum bilirubin, urine urobilinogen and selected liver function tests. If urine urobilinogen is low or absent and the serum bilirubin is stabilized or decreasing, it may be that acholia due to liver failure is present. In such case urgent steps should be taken to relieve the obstruction.

Experimental evidence indicates that acholia occurs when obstruction to the bile passages and infection co-exist. Infection undoubtedly accelerates the cycle in the bile pigment change from pigmented bilirubin through nearly colorless biliverdin. Experimental evidence also has shown that the bile duct epithelium absorbs and excretes, dilutes and depletes the contained bile so that the residual fluid (except for calcium) becomes nearly isotonic with (blood) serum. Hence the bile in the ducts is colorless. Mucin also may be added from intramural duct glands.

Should bile not be formed, then there may be no change in the icterus index. Indeed, it is conceivable that the blood bilirubin level could decrease. This fact may interfere with the prognostic value of repeated determinations of the bilirubin levels.

Evidence of impairment of liver function can be obtained from the following findings:

1. There is a progressive fall in the level of the serum albumin and a rise in the globulin.
2. After an initial rise in the serum alkaline phosphatase there is then a gradual fall.
3. After the initial rise in the levels of serum total cholesterol ester there is an eventual fall.
4. There is no impairment in the ability of the liver to metabolize galactose. Moderate decrease in liver glycogen may be demonstrated.

5 There is a gradual decrease in plasma prothrombin concentration which does not reach hemorrhagic levels
6 There is no change noted in thymol turbidity test

Death from acholia following bile duct obstruction is due to combined liver failure and emaciation

g Hepatic Coma

The patient who becomes unconscious with high fever because of hepatic disease may die because of hepatic insufficiency Frequently sudden hemorrhage from varices because of the co-existent severe hepatic disease will precipitate coma Certain drugs, toxins or chemical poisons which accumulate in the hepatic parenchyma can precipitate hepatic insufficiency Inadequate nutrition, continued intake of alcohol, low sodium and vitamin poor diets are often factors in liver failure

In many cases of chronic liver disease, hepatic coma occurs from failure to maintain normal levels of total fluid and electrolytes and in the presence of infection, hemorrhage and surgery (including liver biopsy or abdominal paracentesis) Any cause for hepatic cell anoxia can increase liver damage Physical overexertion alone, with overproduction of bicarbonates and associated increase in plasma carbon dioxide, are sufficient to depress liver function

Sudden decompression of the common bile duct at surgery may produce shock Obstructed bile capillaries had previously displaced a large volume of circulating blood from the liver With release of back pressure in the bile capillaries, space for circulating blood is again available As a result 500 cc or more blood may be suddenly diverted into the hepatic circulation If there be insufficient reserve the circulating blood volume may be in chaos Unless the circulating blood volume is augmented by plasma or blood or its equivalent there is imminent danger of hypotension or anoxemia, of pulmonary edema and of hepatic failure

A patient unconscious by reason of hepatic insufficiency may have an icteric tint to skin and sclerae may have ascites and/or pleural effusion may have multiple bruises or evidence of hemarthrosis and hepatosplenomegaly may be present High fever pitting edema and malnutrition are frequent Spider angiomata and caput medusae may be present Dilated superficial veins may include those in the hemorrhoidal and saphenofemoral systems

In hepatic coma the pulse is rapid and weak the skin is dry and warm, respirations are rapid and malodorous Foul diarrhea and coffee ground vomitus are common Anemia hypoprothrombinemia and hypoproteinemia (with reversal of albumin globulin ratio) are usual Oliguria is frequent

Hepatic coma is to be differentiated from uremia diabetic acidosis or hypoglycemia alcoholism carbon monoxide poisoning barbiturate and morphine poisoning and hemorrhage from the gastrointestinal tract The low sodium syndrome may complicate the picture

h Genito-urinary

(1) PREOPERATIVE STATES Renal disease may co-exist with biliary tract disease The degree to which this affects the biliary system is problematical Ordinarily azotemia will not disturb hepatobiliary function However nephritis may produce defective hematopoiesis by toxic effect on the bone marrow In addition a renal carbuncle or perinephritic abscess may cause a subhepatic abscess Anomalies calculi, infection tumor or degenerative disease can mutually involve both systems

Right flank pain may be associated with renal or biliary calculi infection, tumors or anomalies and these may co-exist Often, differentiation is not possible Both may require treatment In one instance a painful nephroptosis was present in a patient with biliary colic and a normal cholecystogram At operation after repair of the malposition of the kidney the gallbladder was explored An adenoma of the gallbladder was excised through the renal incision and a cholecystostomy established The patient is well

Certain electrolyte deficiencies of the urinary tract may affect the biliary tract and *vice versa* For example excessive diuresis such as that induced by mercurials given for cardiac disease may inaugurate the low sodium syndrome This or uremia may be clinically similar to hepatic coma

Another example is the patient with a chronic bile fistula the effects of which may be confusing Loss of fluid can initiate renal insufficiency loss of electrolytes may produce altered cell physiology particularly because of the low potassium and the combination of cholangiohepatitis and diminished utilization of food causes hypoproteinemia

Renal disease may follow jaundice by reason of the hyperbilirubinemia and a pigment nephrosis may occur

(2) POSTOPERATIVE STATES Special precautions apply to patients with cardiac and renal disabilities when they are recovering from biliary tract surgery Particular attention must be paid to maintaining an adequate renal output This requires adequate circulating blood volume supportive therapy to the myocardium prevention of dehydration and use of enteral routes for hydration and nutrition whenever possible

The older patient does not require the same large fluid volume as the patient under 50 years Since diabetes affects electrolyte balance and anemia modifies the blood volume both of these must be normalized to avoid additional renal (and cardiac) damage

In the postoperative patient peripheral circulatory failure or a shock like state may occur. This may be due to blood loss and to the extensive dissection, although blood loss may not be obvious. There may be additional postoperative blood loss in the operated area. Anoxia during and following anesthesia may damage renal filtration mechanism. There may also have been fluid loss with excessive respiration, vomiting or ileus.

The kidney may also fail by reason of (a) an incompatible blood transfusion, (b) the presence of severe icterus and (c) the presence of infection. This renal insufficiency is due to obstructed excretion.

Still another cause for renal insufficiency is that due to nephrotoxic medications including mercury, bismuth and such organic materials as uric acid, and can be due to sulfonamide sensitivity, serum sickness and bacitracin.

Prevention is the best means to combat renal insufficiency. Proper hemostasis, blood and fluid volumes, accurate cross matching of blood used for transfusion, adequate oxygen and airways (particularly following anesthesia) are among several precautions. Determining beforehand the patient's response to medications and past experiences with anesthesia and antibiotics may prevent some unexplained abnormal (allergic or hypersensitivity) phenomena which can precipitate renal insufficiency.

The kidney which is functioning normally will usually be able to withstand shock or temporary obstruction. The kidney, however, which is badly damaged as a result of pre existing disease is unable in many cases to return to a normal state.

Patients with incipient or active renal insufficiency require (a) maintenance of blood volume and correction of anemia, (b) accurate replacement of fluid loss, (c) maintenance of blood pressure, (d) adequate oxygenation, (e) oral feedings in lieu of parenteral fluids, (f) use of specific antibiotics and other drugs which are compatible, (g) careful management to prohibit damage from other conditions.

2 Cardiopulmonary Systems with Relation to the *Biliary Tract*

Monroe evaluated the older patients with gallstones at Peter Bent Brigham Hospital. There were 455 patients over 60 years of age *who refused* surgery. Of these 28 died because of stones and 53 others died of diseases which received contributions *from the stones. This is a mortality rate of* 18 per cent, which far exceeds what would be expected from operations. *The most common co-existent factors* were biliary tract and cardiac disease.

Gallbladder disease augments angina and fibrillation. Furthermore attacks of biliary colic and angina may falsely represent each other. Walters, Ravdin and Wolferth have indicated that careful clinical differentiation is required. Mild myocardial ischemia may be augmented by pain from re

current biliary colic to induce myocardial infarction In the patient without myocardial disease biliary colic may present pseudo-anginal symptoms—even to specific electrocardiographic findings

Differentiation between cardiac and biliary origin for pain may not always be made Biliary colic will awaken the patient from sleep angina does not Exertion, except for its deleterious effect in hepatitis and on liver glycogen does not affect the bile ducts, it is more often followed by angina The patient with biliary colic may not have dyspnea or cyanosis The patient with myocardial ischemia rarely has distention and flatulence Significant changes in cardiac rate rhythm and output usually occur only with pre-existing cardiac disease Overdistention of the common bile duct under experimental conditions can produce changes in the R T segments and in the T waves These changes are probably mediated through the vagus fibers (Fitz Hugh and Wolferth)

The cardiac patient may have a non functioning gallbladder with stones That symptoms of gallbladder disease are not recognized does not indicate the absence of deleterious effects

It is not usual for biliary hepatic disease to exert toxic effect upon myocardium or in the cardiovascular system per se However in patients with prolonged jaundice a myocardial depression such as bradycardia may be noted The cause is still not known

Hepatobiliary disease may be followed by pleuritis pneumonitis and atelectasis particularly in the right lower lobe Pulmonary infarction abscess or lobar pneumonia are unusual Traumatic fistulae (gun shot wounds) may become chronic Pulmonary biliary fistulae may also occur following the perforation of a liver or subdiaphragmatic abscess Under these conditions chronicity is usually associated with stricture or other cause for delay in normal bile flow Bile in the sputum is phenomenomally diagnostic of a biliary pleural bronchial fistula

Other causes for pain in the right costal margin and epigastrium simulating either angina or cholecystic disease may be produced by (a) mastitis (b) herpes zoster (c) adiposa dolorosa (Dercum s) (d) intercostal myalgia and neuralgia (e) acute fibrinous pleurisy (f) empyema thoracis (g) pulmonary embolism (h) spontaneous pneumothorax (i) esophageal diverticulum (j) esophageal malignancy (k) metastatic malignancy (l) pericarditis (m) aortic aneurysm

3 Gastrointestinal and Nutritional Facets

Benign and malignant traumatic and inflammatory disease of the gastrointestinal tract may affect the bile ducts Symptoms may refer mutually to and fro affecting or masking the bile ducts

a Pyloroduodenal Dysfunction

Duodenal ulcer hypertrophy of the pyloric sphincter, duodenitis and duodenal diverticula may be associated with disease of the biliary tract Duodenal ulcer and duodenal diverticula may obstruct the extraduodenal common bile duct Pyloric obstruction may cause reflex cholecystic dysfunction Pylorospasm may be a symptom of cholelithiasis Duodenitis may produce (Vater) papillitis which may, in turn cause cholangitis or pancreatitis

Therapy for duodenal ulcer is often difficult in the presence of cholecystic disease The typical Sippy diet is not conducive to satisfactory biliary tract function In addition, hypertrophic gastritis may be caused by the same factors as produce pancreatitis

Furthermore a triad of symptoms may be present cholecystitis (perhaps with calculi), duodenal ulcer and hiatal hernia The recognition of hiatal hernia is often accidental The hiatal hernia may be increased by recurrent vomiting caused by the duodenal or gallbladder disease This triad appears in the older age groups and usually in the chronic ulcer disease patient who has taken much cream over many years

b Intestinal Obstruction

Excluding the *postoperative ileus* which may occur after any laparotomy, the problem of intestinal obstruction is often considered in biliary tract disease *The most direct relationship is in "gallstone obstruction* which follows a cholecystoduodenal (or cholecystocolic) fistula Proven *choledochal enteric fistulae as precursors to gallstone ileus are rare*

Pancreatitis may be severe enough to establish and to mimic mechanical small bowel *obstruction The coincidence of biliary tract disease with pancreatitis* and with intestinal obstruction is not unforeseen therefore

Patients who have had a cholecystoenterostomy are prone to develop small bowel obstruction at the site of the anastomosis unless *a complementary entero-enterostomy* has *been done* Following relief of the biliary tract distention the gallbladder will shrink carrying with it the anastomotic *stoma and causing a kink to the small bowel The stoma may* become inefficient as well

c Appendicitis

In view of the frequency of appendicitis it is not unusual *for it to be* confused with an acute exacerbation of chronic calculous cholecystitis *When this occurs treatment of the gallbladder disease* may be feasible *cholecystostomy* or cholecystectomy may be indicated

Acute cholecystitis may follow appendectomy or, as Schwegman points out, any intra abdominal procedure By reason of the fasting which the

patient undergoes the gallbladder may not empty Codeine or prostigmine may inaugurate biliary tract stasis and augment infection Food taken after the period of starvation may institute biliary tract symptoms

Sequelae of perforated appendicitis may occur as a subhepatic or subphrenic abscess Rarely, a portal pylephlebitis may occur with its sequelae of severe multiple liver abscesses

d Tumor

The duodenal loop is a common site for the appearance of metastatic malignancy from the gastrointestinal tract The lymph nodes, particularly the peripancreatic and the hepatic pedicle groups which surround the portal vein may often be involved It is important to be alert to these since therapy as well as prognosis necessarily alters Symptoms are usually related to the primary malignancy

e Fungi and Parasites

Biliary tract disease may be coincident with or follow fungous or parasitic infection of the intestinal tract The Actinomyces Blastomyces, Nocardia and other fungi have been reported in liver abscess These can involve the hepatic pedicle to produce obstructive jaundice Sarcoidosis usually does not involve the biliary tract except as a result of intrahepatic expansion

Intestinal parasites may lodge in the gallbladder Ascaris Taenia and others may occlude the common or cystic ducts Much of this disease occurs in areas of limited sanitation wherein the systemic effects of the protozoal or parasitic diseases by far surpass any specific manifestations in the biliary tract Hepatic schistosomiasis is only one part, although prominent of a generalized disease

f Nutrition and Electrolytes

Malnutrition and avitaminosis are present frequently in patients with severe biliary tract disease They are the commonest complication of advanced disease of the common bile duct particularly associated with calculi Enzymatic digestive function is depressed In addition since many foods can initiate biliary colic patients with cholelithiasis often will not eat anything Protracted vomiting may occur with its associated loss of chloride and total base Withal liver glycogen is depleted and hepatic cell function deteriorates

The most significant feature of nutritional deficiency associated with biliary tract is that it may develop rapidly without much clinical evidence Hypoproteinemia is often marked by a reversal of the albumin globulin ratio When marked hypo-albuminemia is found the patient is edematous

In addition, a depletion in blood volume will often occur before hypoproteinemia and this before anemia These three abnormalities may appear suddenly The best indication regarding uncompensated blood volume loss, hypoproteinemia and loss of hematopoietic reserve is in the anamnesis regarding restricted diet intake and significant reduction in body weight These may be confirmed by laboratory determination of protein, bromsulfalein excretion and studies of blood and plasma volume

Nutritional deficiency is not always obvious in the diabetic, nephritic or cardiac patient These metabolic disturbances must be evaluated to avoid low sodium syndrome, ketosis, diabetic coma insulin shock, gout, uremia and pulmonary edema All these may be among the more serious coincidences in biliary tract disease

In the patient with chronic biliary fistula the problem of electrolyte replacement depends upon the fistula When only bile is lost, replacement of potassium is important Pigment, bile salts, and cholesterol are not particularly vital Pancreatic fluid loss must be compensated by adequate base and bicarbonates An associated intestinal fistula will also deplete chlorides Empirical replacement may be satisfactory if such losses are the only ones to be considered However, associated renal disease introduces additional factors such as acidosis

Cardiac disease may require limitation of fluids and chlorides

Clinical signs of dehydration edema, cyanosis, mottled skin flush hyperpnea tachypnea or Kussmaul type respirations, oliguria, perspiration or melena demand correction by restoration of normal electrolyte and nutritional patterns The cutaneous and nerve symptoms of avitaminosis are readily identified

The normal individual will attempt to compensate for grievous imbalance, *but homeostasis is lost rapidly in the child and the older patient*

D Other Factors

1 Biliary Tract as Focus for Infection

The gallbladder has been indicated as a focus for infection This is true in many cases of typhoid or colon bacillus infection *Cholecystectomy will* eliminate positive stool cultures of the Salmonella group of bacilli However it has not been positively proven that chronic cholecystitis is a major cause for fasciitis arthritis, neuritis and other manifestations of the "septic focus"

As an example there is the case of Mrs R W aged 49 who had severe progressive deforming arthritis of a mixed type Gallstones and kidney stones were present Urinary symptoms were severe because of a gangrenous pyelonephritis associated with pressure *from an eroding calculus* Nephrectomy was required The patient had evidence of reversal of the albumin globulin ratio with a marked weight loss We might have considered renal

ual renal and active biliary tract disease as responsible for activating the joint symptoms However, because she also had a peculiar skin rash biopsy was taken This indicated the presence of the collagen disease 'dermatomyositis ' Sufficient systemic manifestations were present that despite temporary relief with cortisone the patient died within eight months after the nephrostomy Our experience is that it is unusual for the gallbladder and biliary tract to act as a focus of infection

Relief of general symptoms which follows cholecystectomy is usually due to its salutatory effect on the biliary tract more than to elimination of toxins or bacteria

2 Backache

Many individuals observe residual backache following trauma Backache may result from musculoskeletal imbalance or it may accompany gastrointestinal or genitourinary symptoms Occasionally backache particularly in the left loin may be due to an expanding cystic inflammatory or malignant pancreatic lesion There is nothing specific in the backache which accompanies pancreatic disease to permit its early rapid identification Diagnosis thereof is often made by exclusion after clinical consultative and radiologic studies When symptoms warrant it exploratory laparotomy is done Backache per se is not sufficient to justify laparotomy

3 Pruritus

Just as is backache so is pruritus a symptom recognized as being produced by pancreato biliary tract disease In itself pruritus does not indict the liver or bile ducts However itching accompanies diabetes and is usually present in chronic obstructive jaundice Its mechanism in jaundice is presumed to depend upon nerve irritation by retained bile salt rather than bilirubin products Itching may be severe enough to induce a severe factitial dermatitis insomnia and suicide It may continue after the bile obstruction has been relieved

Many agents have been used for relief of the intolerable itching local lotions and creams may be eminently successful Procaine amide (Pronestyl) hydrochloride ergotamine androgens calomel and intravenous 0 1 per cent procaine have all been utilized A most encouraging report recently recommended the use of 25 mg daily of sublingual methyltestosterone (indicating a possible interrelationship with internal cholesterol metabolism) (Lloyd Thomas)

4 Excessive Perspiration

Fibrocystic disease of the pancreas affects the pulmonary and integumentary systems Emphysema and pneumonia occur frequently in these patients In addition marked sweating and a marked increase in salt loss

occur in late stages of pancreatic deficiency Salt depletion with excessive sweating may be occasionally considered as indicative of decreased pancreatic function

Sweating in patients with migrating phlebitis is on a localized basis whereas salt and fluid loss due to excessive perspiration in pancreatic disease is a generalized phenomenon

5 Thrombosis and Phlebitis

Migrating superficial phlebitis, *as manifest by localized yet migrating* pain and redness and increased localized cutaneous moisture, can be due to pancreatic disease It frequently appears as thrombosis secondary to venous incompetency The exact etiology of this migrating phlebitis and thrombosis is not understood Many investigators feel that disturbances in the trypsin antitrypsin ferments are responsible Pressure by the pancreas on the caval system and on other major veins may be a factor in the edema and phlebitis

One of the early clinical symptoms of pancreatic disease is the history *of recurring migrating superficial phlebitis*

6 Bile Duct Obstruction without Icterus

Jaundice may not always appear when the bile duct is obstructed In a few individuals the serum bilirubin will be only slightly above normal levels even though the bile duct is obstructed The urine and fecal urobilinogen however, are absent and suggest the diagnosis

At operation the common duct bile is grayish green with green brown mud and sludge Calculi are found together with a mild or moderate cholangitis In such cases bile flow may cease for four or more hours postoperatively The bile thereafter is green, thick, normal For a few days thereafter an icteric tint may appear in the sclerae and serum bilirubin is increased

Liver biopsies taken under similar circumstances have revealed "*cholestasis*" (Popper) In these patients, however, regurgitation or other mode of entrance of the bile pigment into the serum *does not* occur until after release of the obstruction The additional pigment is picked up then from the periportal spaces and hepatic cells because of the improved hepatic circulation It is presumed that either adequate blood circulation is denied to the area of pigment deposition or that this form of bilirubin is a nonabsorbable fraction

7 Hyperemesis Gravidarum

Nausea and vomiting which occur during pregnancy may be due to organic, endocrine or psychologic aberrations Among organic causes for hyperemesis gravidarum is acute cholecystitis In certain cases the diagnosis

of cholecystitis may be suspected Accurate statistics are not immediately available but it is suspected that cholecystitis is more common than appendicitis during pregnancy

Flatulence food provocation and jaundice are uncommon in biliary tract disease during pregnancy whereas nausea, vomiting and right flank or epigastric discomfort are frequent Use of cholografin by intravenous route is a valuable method of differentiating cholelithiasis from other causes for vomiting during pregnancy During an interval of freedom from nausea and vomiting there is no objection to the use of oral cholecystography for diagnosis in a patient with hyperemesis gravidarum

8 Mental Reactions

Anxiety and apprehension are normal preoperative mental reactions However depression and melancholy are often seen additionally in patients with severe hepatic disease The ' bilious cantankerous and vituperative ' patient who often vents his spleen on his surroundings may only have severe biliary tract disease

Postoperative psychoses are no more frequent following biliary tract disease than other surgical procedures They do occur Severe anxiety-depression psychoses are the most common syndromes Prognosis depends upon the degree of mental illness and method of therapy adopted In many patients shock therapy (electro-shock or insulin) cannot be utilized in the immediate postoperative period

The patient with malignancy of the pancreatobiliary tract may survive with metastases for many years The patient should not be told of his malignancy Heirs and next of kin however should be given all details The patient can be advised that a possible calamity may occur and such representation made to him as if it were a routine statement for all patients Exceptions are customary since individualization in management is essential

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7

LABORATORY OBSERVATIONS

A General

Many aspects of liver diseases and jaundice remain unsolved and differential diagnosis may be made more readily on the basis of clinical observation than as a result of laboratory studies. This is because available liver function tests cannot measure a unit function of the liver. The hepatic cell can perform multiple functions and the descending order of failure is individually variable. Hepatic disease does not affect cellular function uniformly because several pathological processes may be present to influence results or tests in various and occasionally inconsistent ways. Much of an otherwise diseased liver, also may have normal function.

Liver biopsy can correlate clinical, functional, structural and laboratory findings. However, since biochemical methods are less uncomfortable and are universally available they are preferred to biopsy.

B Liver Function Tests

Although the liver excretes and affects essential metabolic changes liver insufficiency may exist without clinical symptoms. Laboratory tests as a single isolated procedure may be misleading. Liver function studies are best performed serially and in groups.

The degree of liver cell damage is statistically correlated with an increase in cephalin flocculation, thymol turbidity and total and direct bilirubin and with a decrease in albumin and albumin globulin ratio. Elevated values of alkaline phosphatase and urine urobilinogen are associated with the presence of liver cell disease but not with its degree. Total serum proteins, non protein nitrogen, fecal urobilinogen, prothrombin percentage and serum cholesterol (including esterified fraction) reveal no significant correlation with degree of liver damage.

1 Serum Bilirubin

It is probable that all or nearly all bilirubin is bound to serum protein. The direct reacting bilirubin is loosely bound to albumin while a more stable complex is formed with globulin. The immediate or delayed development of color in the Van den Bergh reaction is dependent upon the diazoti

zation rate not upon variable mixtures of two different forms of bilirubin The one minute total bilirubin ratio tends to be low in hemolytic jaundice

2 Bilirubinuria

Renal threshold for bilirubin varies in accordance with function of renal epithelium and based upon antecedent renal disease It is significant that bilirubinuria may be encountered early in hepatitis or obstructive lesions when the total serum bilirubin is within normal limits but the one minute bilirubin is elevated During the recovery period bile pigments disappear from the urine when one minute serum bilirubin drops to 0 8 to 1 2 mg per cent Interrelationships between hepatic enzymes and renal cell filtration are undoubtedly active

3 Urobilinogen

When bilirubin enters the intestine bacteria convert it to urobilinogen urobilin and stercobilin Normally some of the urobilinogen is absorbed and returned to the liver The hepatic cell may excrete it again or the Kupffer cell may convert it to hemoglobin Whichever occurs there is little or no urobilinogen remaining in the blood that leaves the liver Small amounts appear in the urine (0–4 mg in 24 hours) Fecal urobilinogen varies from 40 to 280 mg in 24 hours

In jaundice due to hemolysis urine urobilinogen is normal but fecal urobilinogen may be increased If marked suppression of bile excretion occurs it must be evident that little or no urobilinogen will be formed and consequently little or none will be absorbed This fact must be kept in mind in the interpretation of low urine urobilinogen levels when other function tests indicate hepatic insufficiency In such cases the reappearance of urobilinogen and its rise above normal indicates return of bile excretion If little or no bile enters the intestine little or no urobilinogen can be formed (Plate One)

When antibiotics are employed the suppression of bacterial enzyme activity may prohibit the conversion of bilirubin to urobilinogen even though hepatic function may be normal This is not always present

4 Serum Proteins

The liver synthesizes the major portion of serum albumin Globulin probably is produced by the mesenchymal (Kupffer) cells Paper chromatography and paper electrophoresis (or iontophoresis) will give much new information

In hepatitis albumin values fall and gamma globulin values rise

Hypoproteinemia is frequently found in the jaundice of both portal and biliary cirrhosis Hypo-albuminemia is common Patients with hepatic

insufficiency usually show diminution of albumin *with elevation of gamma* globulin

5 Flocculation tests

Cephalin cholesterol flocculation is related to the elevation of gamma globulin and the diminution of serum albumin

Thymol turbidity reaction is related to the beta globulin lipid complex About half of the patients with biliary cirrhosis react abnormally regardless of the absence of jaundice

Zinc sulfate turbidity is related to gamma globulin

6 Total Cholesterol and Cholesterol Esters

The ratio of free to total cholesterol rises in both obstructive jaundice and hepatitis *It is highest early in the icteric phase of infectious hepatitis* and in chronic organic bile duct obstruction Portal cirrhosis demonstrates an elevation of total cholesterol sometimes reaching 1000 mg per 100 cc of serum *It is generally agreed that a low cholesterol ester ratio occurs in* cases of hepatocellular disease The liver cell is the seat of esterification and therefore liver cell injury interferes with this function

Esters are significantly lowered in some cases of obstructive jaundice when only serum bilirubin and alkaline phosphatase are elevated A low ester value and moderately high serum bilirubin when other function tests are normal are more likely due to extra hepatic biliary obstruction than to primary hepatocellular disease

7 Serum Alkaline Phosphatase

Commonly hemolytic jaundice is characterized by normal serum alkaline phosphatase Cases of hepatitis show a moderate elevation, usually less than 10 to 15 Bodansky units This is also true in portal cirrhosis Biliary *cirrhosis shows considerably higher values In extrahepatic biliary obstruc*tion values are usually over 10 units

Serum alkaline phosphatase is formed by the liver cell Phosphatase may be slightly elevated in hepatitis Serum phosphatase is also increased in a variety of skeletal disorders

8 Bromsulfalein

This dye is (apparently) excreted by the liver It is customary now to determine the degree of retention at the end of 45 minutes Bromsulfalein is retained both in obstructive lesions and in liver cell disease Circulatory *impairment may also cause retention without jaundice* In hepatitis and cases of obstructive jaundice some dye may persist in the circulation as

long as 30 days It is advisable to pretest sera whenever the bromsulfalein test is repeated on the same case

9 Prothrombin Activity

The activity of prothrombin before and after the administration of vitamin K is of some value in differential diagnosis Failure to obtain a distinct rise after vitamin K is given in adequate dosage is highly suggestive of hepatic insufficiency

10 Serum Cholinesterase

In the presence of liver cell disease serum cholinesterase values fall Serum cholinesterase is of value in separating cases of portal cirrhosis without ascites from the remainder of the group of hepatobiliary diseases Benign obstructive jaundice viral hepatitis abdominal malignancy and cachexia also are characterized by low values

11 Liver Test Profile

In order to obtain maximum information about the pancreato-biliary tract many physicians will order a battery of liver and pancreatic function tests Depending upon the individual laboratory the physician and the patient these may be of increasing significance as they are repeated and studied in orderly fashion

As a suggested group the following may act as a liver pancreas profile (a) bilirubin prompt direct delayed direct and total (b) cephalin cholesterol flocculation (c) colloidal gold (d) thymol turbidity test—18-hour factor (e) bromsulfalein retention—2 mg (f) inorganic phosphorus (g) alkaline phosphatase (h) total fatty acids (i) neutral fat (or total lipids) (j) total protein including albumin and globulin (k) amylase (l) lipase (m) urine bilirubin and urobilinogen and (n) prothrombin time (Fibrinogen and electrophoretic studies of the proteins will be increasingly important)

C Laboratory Aid in Diagnosis of Jaundice

Jaundice may be due to interference of bile flow it may occur when bile passages are unobstructed (Plate One)

Jaundice without impairment to bile flow may result from overproduction of bilirubin (hemolytic) It may also be due to an elevation of the excretion threshold (familial non hemolytic) Clinical and laboratory diagnosis of these is not difficult Recognition is accomplished by the absence of bilirubinuria by a negative Van den Bergh reaction of the serum bilirubin and by hematologic surveys Tests for spherocytes sickling and fragility should be done

Obstruction to bile flow may be intrahepatic or extrahepatic. This differential diagnosis may not always be made by either clinical or laboratory methods. Therapy in "surgical jaundice" is clear cut if some obstructive mechanism be identified, such as tumor, stone or stricture. But the same picture is presented by the so-called "medical jaundice" which appears in hepatitis and cirrhosis. As a rule, intrahepatic jaundice is a complication of hepatitis or cirrhosis which appears as liver cell degeneration. Extrahepatic obstruction produces liver cell necrosis secondary to extravasation of bile from extremely dilated bile ducts into the lobular periphery ("cholestasis" as described by Popper).

Liver cell degeneration is prominent in jaundice requiring medical treatment (hepatitis and cirrhosis). In the jaundice which requires surgical treatment bile pigment is in Kupffer cells and the dilated bile capillaries contain inspissated bile. Bile pigment is also seen in the liver cells but little or no cellular inflammation or degeneration is present.

Flocculation tests and the alkaline phosphatase determination are most useful in distinguishing medical from surgical jaundice. However, intrahepatic and extrahepatic obstructive jaundice cannot always be differentiated (cf. chapter 6). In addition, should severe bacterial invasion be superimposed upon extrahepatic biliary tract obstruction, the laboratory picture is confused. As a result of the infection, serum gamma globulin levels are altered and flocculation tests become unreliable.

D. Microscopic Examination of Bile, Including Bacteriology, Immunology and Cytology

Bile can be obtained through the duodenal tube. In normal cases the first color is lemon to golden yellow. A deep golden yellow, nearly green, thick syrupy color may be seen next. After this, a combination of the first two can be drained. The first bile is from the common duct; it usually consists of about 5 to 8 cc. The second bile originates in the gallbladder; there may be 20 to 30 cc. in this fraction. The third type is a mixture of duct and gallbladder bile and may range from 10 to 30 cc.

(Schalm (1953) has suggested a special technique he calls "bilitinction." After emptying the gallbladder, Bromsulfalein (200 mg.) is injected. The duodenum is intubated and the bile sample can colorimetrically be identified as hepatic or gallbladder bile.)

Variations in the character of bile may suggest a diagnosis. Absence of dark concentrated bile may indicate disease in the gallbladder or a postcholecystectomy state. Absence of bile may confirm an obstructive lesion whereas in the presence of jaundice the appearance of bile may suggest relief of the obstruction or the presence of "medical" jaundice. In cases of

post cholecystectomy syndrome duodenal intubation is reinforced by use of the secretin test (vide infra) to evaluate pancreatic function

Blood which appears during duodenal aspiration may be factitial or significant Additional observations can be done and studies do not necessarily have to be discontinued when blood appears

Even without elaborate precautions for sterility it is feasible to examine smears for bacterial organisms It is also possible to isolate pathogenic invaders and to establish sensitivity to antibiotics

Using accepted immunologic methods bile fractions may be studied for antibody content after chemical sterilization and Seitz filtration Streptococcus and colon bacillus agglutinins are frequently present

Bile and duodenal contents may be prepared for cytologic examination by staining the sediment It is possible to recognize exfoliated epithelial cells arising from the pancreas the bile ducts the gallbladder the liver and the duodenum

In obtaining fluid intended for cytologic examination the aspirated gastric juice and bile fractions are collected in centrifuge bottles or receptacles chilled in ice The fluid is neutralized to pH 7 and centrifuged at 1 800 revolutions per minute for ten minutes Sediment is used for making smears and these are fixed while moist for one half hour in chilled acetone They are then dipped in 1 per cent celloidin in acetone drained for ten seconds and then transferred to 95 per cent alcohol Staining process is then accomplished in accordance with the Papanicolaou technique (Lemon and Byrnes)

Normal duodenal aspirate contains few cells and a little mucus Calcium bilirubinate and cholesterol crystals may appear the latter is suggestive of lithiasis Inflammation in the bile ducts may be indicated by pus cells macrophages the appearance of columnar epithelium or excess amounts of pigment or crystals

Malignant cells when present are usually accompanied by groups of normal columnar epithelium Malignant cells are not arranged in a mosaic they are irregularly grouped Nuclei in malignant cells vary widely in size shape and staining characteristics The nuclei are usually larger with relative increased proportion to the cytoplasm Nucleoli may be present Cytoplasm is basophilic and vacuolated Mitoses are not usually seen

Similar studies may be performed on bile obtained at operation or postoperatively by drainage from the gallbladder or ducts

Chemical bacteriologic cytologic and immunologic studies should be done as an auxiliary mode for diagnosis of hepatic function to exclude malignancies and to identify bacterial organisms The common bacterial invaders of the biliary tract include *Escherichia coli* *Aerobacter aerogenes* and *Streptococcus fecalis* Proteus pseudomonas Corynebacterium and

anaerobic organisms have been identified. Bacteria are probably present in the gallbladder or bile of 50 to 70 per cent of patients with chronic choledocholithiasis.

I Examination of Intestinal Content for Pancreatic Ferments and Other Tests of Pancreatic Function

Duodenal intubation to obtain pancreatic secretion was developed by Einhorn. He indicated normal values for amylase (60 mm.), lipase (4 mm.) and trypsin (3 mm.). The normal rate of flow is 1.0 cc. per minute. Abnormalities may be indicated as euchylia, hypochylia and hyperchylia as related to quantity of secretion or as hyper and hypopancreatism regarding quality of fermentation.

The secretin test of pancreatic function is very valuable. Following its injection there is an increase in flow of pancreatic juice up to 3 cc. per minute, with a proportionate increase in enzyme content. When a neoplasm obstructs the pancreatic duct, the volume of juice is diminished. Enzyme concentration, however, is not altered in response to secretin. In patients with fibrosis or calcification of the pancreas, the volume of juice may be slightly increased but enzyme content is deficient.

The value of serum amylase and lipase determination is known to increase under certain conditions due to pancreatic disease. It should be remembered that mumps, renal failure, peritonitis and narcotics can all cause an elevation in the serum amylase. Serum lipase is slower to rise and delayed in its fall in patients with acute pancreatitis.

Antithrombin titer may be significantly elevated in inflammatory pancreatic disease. Another test for pancreatic function is the glycine utilization (conversion from gelatin) which may serve to identify pancreatic fibrosis. Normal pancreas function will reveal a rise in plasma glycine to 2.5 times normal within two to three hours after ingesting gelatine. Any impairment in pancreatic function, such as a fibrosis, is not associated with this rise.

Stool examination is of considerable importance in evaluation of patient with pancreatico-biliary disease. Although considerable variation in content is normal—character, color, frequency and reactions to tests for bile, urobilinogen and blood are of great value.

Creatorrhea is seen in pancreatic disease in that 50 per cent or more of ingested meat fibers appear in the stool. Normally 90 per cent is utilized. Microscopically, in such cases, deficient trypsin is noted by the appearance of many meat nuclei and fibers. Absence of creatorrhea is not significant.

Steatorrhea is of great diagnostic significance. Macroscopically the stool is white and has an aluminum metallic luster. Feces are rancid and foul and may be greasy or solid. Fat crystals are seen. Coincidentally, amylorrhea due to unexploded starch granules may appear.

Laboratory Findings Pancreato Biliary Disease

I Urinary Tract Findings In gross examination of the urine specimen, its frothiness and burgundy wine color may be sufficient to identify bile However of great importance are serial daily determinations of urinary urobilinogen Both of these tests may be bedside procedures

Presence of bilirubin and urobilinogen at the same time are indicators that jaundice is probably medical ' However serial measurements of trends in level is of real significance The urinary bilirubin should decrease as the urobilinogen is increasing if the patient is recovering from an episode of calculous obstruction to the common bile duct Should bilirubinemia or bilirubinuria decrease without the appearance and increase in quantity of urobilinogen then it is suggestive of hepatic insufficiency

Amylase in the urine is decreased in both pancreatic disease and in nephritis It is increased when amylasemia is present Amylase will remain in the urine for at least 24 to 36 hours after serum levels have returned to normal Lipase and trypsin determinations in the urine are not significant nor specifically diagnostic since these originate in any tissues of the body

G Laboratory Data

1 Milliequivalents Milliequivalents measure chemical relationship between various ions One m Eq of sodium is chemically similar to one m Eq of any substance although their actual weights differ in milligrams

The equivalent weight of a substance is a chemical term defining that weight of the substance which combines with or displaces one gram atom of hydrogen A m Eq weight of a substance is its equivalent weight divided by 1000 Concentration expressed in mg per 100 cc is changed to m Eq by multiplying it by 10 to bring 100 cc to a liter and then dividing by the equivalent weight of specific ion The equivalent weight of the monovalent ions is equal to the atomic weight

2 Specific Laboratory Data

Conversion of electrolyte values to m Eq /l (table 12)

Normal ranges in serum (plasma) of electrolytes in m Eq /l and mg / 100 cc (table 13)

Electrolyte concentrations of commonly used parenteral fluids (table 14)

Serum (blood plasma) normal levels table 15)

Bile—normal levels (table 16)

Pancreatic fluid—normal levels (table 17)

Urine—normal levels (table 18)

Feces—normal levels (table 19)

TABLE 12 CONVERSION OF ELECTROLYTE VALUES TO mEQ/L

Electrolyte	Atomic Weight	Valence	Equiv Weight	Multiply by	Divide by
Sodium	23	1	23	0.434	2.3
Potassium	39	1	39	0.256	3.9
Calcium	40	2	20	0.500	2
Magnesium	24	2	12	0.833	[illegible]
Bicarbonate	—	—	22.4	0.446	[illegible]
Chloride	35.5	1	35.5	0.281	3.5
Phosphate inorganic	31	1.8	17.2	0.580	[illegible]
Sulfate inorganic	32	2	16	0.625	1.6
Protein	—	—	—	2.43	

TABLE 13 NORMAL RANGES IN SERUM (PLASMA) OF ELECTROLYTES IN mEQ/L AND MG/100 CC

Mg/100 Cc	Electrolyte	mEq/L
310–340	Na	135–147
16–22	K	4.1–5.7
9.0–11.5	Ca	4.5–6.0
4.25–5.25	Mg	2.1–[illegible]6
55–70	CO	25–31
45–60	O	20–26
[illegible] Per Cent		mM/L
350–375	Cl	98–106
4–[illegible]	PO_4	2.[illegible]–4.5
0.5–2.5	SO_4	0.3–1.5
6–8	Prot	14.6–19.4

Table 14. Electrolyte Concentrations of Commonly Used Parenteral Fluids

Solution	Gm/L	mEq/L Cations	mEq/L Anions	Calories
Isotonic saline (0.9% NaCl)	9.0	154 Na	154 Cl	0
Hypotonic saline (0.45% NaCl)	4.5	77 Na	77 Cl	0
Hypertonic saline (5.0% NaCl)	50.0	850 Na	850 Cl	0
Ringer's solution (isotonic solution of three chlorides)	8.6 NaCl 0.3 KCl 0.33 $CaCl_2$	147 Na 4 K 6 Ca	157 Cl	
One-sixth molar lactate (molar solution diluted 1 to 6)	18.66 Na lact	167 Na	162 mM lact	48
Sodium bicarbonate (1.5%)	15.0 $NaHCO_3$	178 Na	178 mM bicarbonate	0
Ringer's lactate	6.0 NaCl 0.3 KCl 0.2 CaCl 3.1 Na lact	130 Na 4 K 4 Ca	111 Cl 27 mM lactate	10
5% glucose	50.0 CHO			200

TABLE 15 SERUM (BLOOD PLASMA) NORMAL LEVELS

Determination (Blood)	Normal Level
Amylase (serum)	15–100 (Somogyi) units 70–240 mg %
Basal metabolic rate	−10 to +10
Bilirubin (cf "Van den Bergh")	
Bleeding time	1–3 min
Blood volume	Adult—4500 cc (3000 to 7000) Female—45–85 cc/kg body wt Male—66–100 cc/kg body wt
Bromsulfalein excretion	Less than 4% in 45–60 minutes
Calcium (serum)	Total 4.5–5.5 mEq/l (9–11 mg %) Diffusible 2.5–3.0 mEq/l (4.5–6.0 mg %)
Carbon dioxide combining power	Adult 25–35 mEq/l (55–75 vols %) Child 20–30 mEq/l (45–65 vols %)
Cephalin cholesterol flocculation	0
Chloride	100–105 mEq/l (350–390 mg %)
Cholesterol	Total 150–250 mg % Ester 60–140 mg % Ester fraction 50–70%
Cholinesterase	0.68 Δ pH to 1.37 Δ pH per hour
Clotting time	3–10 min
Fragility	0.45–0.30% sodium chloride (hemolysis)
Glucose	70–120 mg %
Glucose tolerance	Following 100 g (orally) a. maximal rise in 90 minutes—200 mg b. return to normal after 120 minutes c. subnormal after 180 minutes
Glycine tolerance	2.5 increase in plasma level (2.5 hours)
Hemoglobin	14–16 gm %
Icterus index	0–10 units
Lipase	0.5–1.5 units

TABLE 15.—*Continued*

Determination (Blood)	Normal Level
Lipoids	Total 570–820 mg. % Neutral 0–200 mg. % Fatty acids 190–420 mg. % Phosphorus 175–330 mg. %
Magnesium	1.6–2.5 m.Eq./L. (1.8–3.6 mg. %)
Non-protein nitrogen	15–35 mg. %
pH	Arterial—7.30–7.49 Venous—7.33–7.52
Phosphorus	Total 8–14 mg. % Inorganic 0.5–1.0 m.Eq./L. (2–5 mg. %)
Phosphatase	Acid 0.5–3.5 King-Armstrong units 0.1–1.1 Shinowara units Alkaline 4–13 King-Armstrong units 2–4 Bodansky units
Potassium	4.1–5.6 m.Eq./L. (16–22 mg. %)
Proteins	Total 16 m.Eq./L. (6.0–8.0 gm. %) Albumin (4.5–5.5 gm. %) Globulin (1.5–3.0 gm. %) A/G ratio 1.4–2.2:1 Fibrinogen (0.2–0.6 gm. %)
Prothrombin	100% of normal control sample time
Sodium	139–152 m.Eq./L. (315–330 mg. %)
Sulphur	0.13–0.17 m.Eq./L. (0.5–1.1 mg. %)
Thymol flocculation	0–1
Thymol turbidity	0–3.5
Urea nitrogen	10–20 mg. %
Van den Bergh (bilirubin)	Direct 0.1–0.2 mg. % Prompt total 0.2–0.8 mg. %
Vital capacity	Female 4000 cc. Male 5700 cc.
Vitamin A	115 I.U.

TABLE 16 BILE (NORMAL LEVELS)

Determination (Bile)	Normal Level Duodenal Drainage	"T" Tube Drainage	Gallbladder
Volume	15–60 cc	150–500 cc /24 hrs	30–100 cc
Appearance	Yellow green	Golden brown	Green
Turbidity	Moderate	None	
Floccules	Occasional	None	None
Viscidity	Thin	Thin	Thick
Microscopy	food particles pigment debris (desquamated epithelium)	Occ pigment or cholesterol crystals	fat globules cholesterol crystals pigment
Calcium (m Eq /L)	16	5–10	5–31
Chloride (m Eq /L)	57	50	50–60
Sod cholate (bile salt) (mg %)	2800	250	100–1600
Cholesterol (mg %) (no ester is present)	300	35	200–400
Phosphorus (m Eq /L)	—	—	40–180
Amylase (units)	65	—	—
Lipase (units)	3–5	—	—
Trypsin (units)	2 8	—	—

TABLE 17 PANCREATIC FLUID—NORMAL LEVELS

Determination	Level
pH	8 3 8 6
Sp gr	1 015
Proteins	0 8 gm %
Albumin	0 5 gm %
Globulin	0 3 gm %
Calcium	0 4–4 7 mg %
Chloride	35–95 m Eq /L
Bicarbonate	30 74 m Eq /L
Sodium	134–142 m Eq /L
Potassium	4 7–5 4 m Eq /L
Amylase	85–310 units
Lipase	300 [illegible]00 units

TABLE 18 URINE—NORMAL LEVELS

Determination	Normal Level (24 Hr Spec F[illegible] Noted)
Water	1000–3000 cc
Solids	55–70 gm
Sp gr	1.005–1.030
pH	4.5–8
Urea	25–35 gm
Creatine	0–0.06 gm
Sodium	3.0–5.0 gm
Potassium	1.5–2.5 gm
Calcium	0.1–0.3 gm
Chloride	10–15 gm
Phosphate	2.5–3.5 gm
Galactose tolerance	After 40 gm (orally) less than 3 gm in urine (6 hours)
Hippuric acid conjugation	(IV) 0.7–1.5 gm benzoic acid in 1 hour (O) 3.0–3.5 gm benzoic acid in 4 hours
Glomerular filtration rate (GFR) (inulin or mannitol clearance)	110–140 cc per minute
Renal plasma flow (RPF) (diodrast or p Amino hippurate clearances)	500–800 cc per minute
Urea clearance	40–65 cc per minute
P S P excretion	40–60% 1 hour 60–85% 2 hours
Dilution Concentration	1.004–1.025
Bile	0
Urobilin	10–130 mg
Urobilinogen	Pos in 1:20 or 1:40 dilution 0–4 mg /24 hours

TABLE 19 FECES—NORMAL LEVELS

Determinant	Normal Level (% of dried weight)
Total fats	4 grams 15–25%
Unsaponified fats	10–15%
Neutral fat	1–2%
Free fatty acid	9–13
Combined fatty acid	10–15
Nitrogen	2.5 grams
Urobilinogen	40–280 mg /24 hours

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8

RADIOLOGY

A General

Diagnosis and therapy in patients with biliary tract disease have been greatly improved by the use of radiologic methods The scout film cholecystography (pre) operative cholangiogram and postoperative cholangiogram are available The surgeon may palpate yet not visualize certain blind spots within the liver ducts or at the papilla It is for such that cholangiography is of inestimable value

Gallstones were studied by x ray about 50 years ago Gilbert Fournier and Oudin (1897) found that cholesterol calculi were not evident whereas pigment calculi were demonstrable on radiography Additional studies over the years by many workers including Neusser Buxbaum Eder and Valenta (Buckstein) improved the technique

Shadows appear on the radiograph in the gallbladder region as fecoliths or other intestinal content renal calculi calcified lymph nodes calcific deposits in the ribs aneurysms or vascular markings These are not easily differentiated from biliary and intrahepatic calculi on the scout film Accordingly radiographic visualization of the biliary tract is essential The use of radio-opaque dyes to demonstrate the biliary tract was first used by Graham and Cole in 1924 based upon preliminary fundamental work of Abel and Rowntree in 1909 The first intravenous studies in the intact patient utilized bromine (tetrabromphenolphthalein) and then iodine (tetraiodophenolphthalein) These were used orally later with greater safety

Radio-opaque media now used are 1) Priodax (β(4 oxy 3 5-diiodophenyl) α phenylpropionic acid) 2) Monophen (4 hydroxy 3 5 diiodobenzyl) cyclo hexane carboxylic acid) and 3) Telepaque (3 amino 2 4 6 triiodophenyl) 2 ethyl propanoic acid)

During the past three to four years cholografin (20 per cent solution of sodium salt of N N adipic-di (3 amino-2 4 6 triiodo-benzoic acid) has been used intravenously successfully to visualize the gallbladder and bile ducts

Postoperative cholangiography or injection of biliary fistulae has been done by one method or another since 1918 The first materials used for study of the biliary fistulae were pastes which contained barium bismuth iodized oil sodium bromide and potassium iodide These materials usually

produced cholangitis, duct obstruction and frequently severe systemic reactions

Since 1930 Diodrast (iodopyracet), Lipiodal (iodized poppy seed oil) and Urokon (sodium acetrizoate) have been used in operative and postoperative cholangiograms with practically no complications It has been just a short step to convert the postoperative to operative cholangiography

There are several clinics throughout the world which utilize direct injection methods for visualization in the intact patient of the gallbladder and bile ducts It is possible under fluoroscopic control to inject the gallbladder with radio opaque dyes It is also feasible to inject the gallbladder at peritoneoscopy These will probably not be necessary now that the common duct and gallbladder can be visualized directly by Cholografin

It may not be long before the following simultaneous diagnostic procedures can be done in the selected case (a) Cholografin visualization of biliary tract (and perhaps kidneys, (b) Diodrast visualization of the spleen portal system and its tributaries, (c) aortography with visualization of the superior mesenteric, celiac axis and renal arterial trees, (d) retroperitoneal air injection to identify the pancreatic outline and (e) barium visualization of the duodenum by means of an indwelling Levine tube

B Preliminary X Ray Studies

On plain film study it is possible to see about 20 per cent of gallstones High fat content in a large non opaque cholesterol calculus may be revealed as a radiolucent shadow A non opaque calculus may be inferentially located For example shadows in the gallbladder region may be seen together with mechanical obstruction to the bowel Careful examination may visualize gallstone impaction at the most distal location of the intestinal gas

An occasional case of gaseous cholecystitis (emphysema of the gallbladder) due to anerobic bacteria may be observed Stolz (1901) described gas in the biliary tract associated with colon bacilli infection Kirchmayer (1925) observed Welch bacillus infection in a swollen edematous gallbladder which he removed Gas in the gallbladder and pericholecystic region is identifiable by x ray (Hegner Stevenson, McCorkle and Fong) Air or gas in the biliary duct system may also be seen due to a fistula between the biliary tract and an adjacent viscus The source for the fistula may be further demonstrated after barium by mouth or enema

Even in the jaundiced patient, cholecystogram and barium studies are of great significance The cholecystogram is done first If very extensive liver damage is present there is failure of gallbladder visualization However, since Rudisill Foley Foot and Carr demonstrated the innocuous nature of the dye its value is immeasurable in differentiating toxic from obstructive jaundice in more than 70 per cent of such problems The intra

venous use of a safe dye such as Cholografin for visualization of the bile ducts and gallbladder is expected to aid differential diagnosis of jaundice (fig 42) Absence of bile duct visualization in jaundice may not be significant

The use of any means to visualize the bile ducts coincident with barium studies of the pyloroduodenum is important A dilated common bile duct may produce a deformity in the post bulbar region or in the bulb itself The recognition of tumors of the pancreatoduodenal region depends upon the distorted barium profile which may hold the key to the diagnosis (fig 40)

Widening of the duodenal sweep and Frostberg s inverted 3 deformities of the duodenal loop are recognizable as evidences of pancreatoduodenal or papillary tumors In addition changes in motor function of the stomach alterations in mucosal pattern of the duodenum and secondary manifestations due to the dilated common duct are evidence of tumor Diagrams (fig 41) serve to emphasize the radiologist s observations (a) mucosal changes in the descending duodenum, (b) the circumscribed filling defect in the gastric silhouette described by Case as the 'pad sign (fig 41), (c) a post bulbar duodenal impression due to a dilated common bile duct (d) the inverted 3' sign (e) diverticulum of the duodenum (f) extrinsic pressure by pancreatic mass on the pyloroduodenal region (g) infiltration and obstruction to the duodenum



FIG 40 TUMOR OF PAPILLA

An intraduodenal mass is recognized as a subtraction defect in the lower portion of the descending duodenum—as a comma shaped shadow Papillary tumors (a) are very difficult to differentiate from normal mucosa (b)

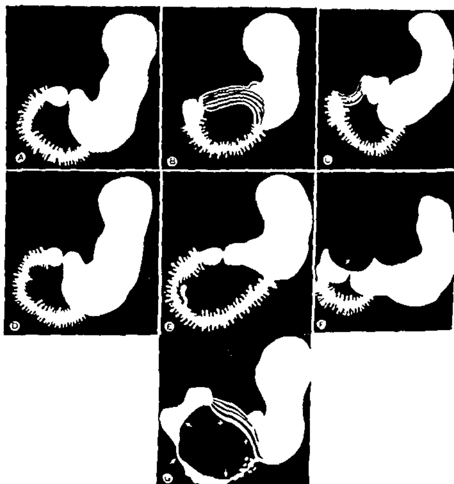


FIG 41 DIAGRAMMATIC DEMONSTRATION OF RADIOLOGIC CHANGES IN STOMACH AND DUODENUM ASSOCIATED WITH LESIONS OF THE PAPILLA AND SURROUNDING TISSUE

Courtesy of *Radiology* and I J Hodes I I Pendergrass and N J Winston

A Early changes include short thick mucosal folds in the duodenum Relative stasis and fixation present

B Case's pad sign is due to extrinsic pressure

C Duodenal impression can be caused by a dilated common bile duct

D Frostberg's inverted 3 sign is also identified as a stopper like protrusion to the left This can be produced by tumor infection or edema of the pancreatic head

E Duodenal diverticulum located near papilla

F Extrinsic pressure on pyloroduodenal region at x may be caused by a mass in the head of the pancreas

G Combined effects of mass and invasion are demonstrated by the pad sign (at X) and by infiltration of in obstructed and displaced duodenum (the arrows)

Calcification in the gallbladder wall or the so-called milk of calcium bile may be visible on the flat plate of the abdomen So-called 'limey bile may be visualized on x ray as a dense calcium shadow with changes in fluid level as the patient changes in position

C Preoperative Cholecystogram

Cholecystography should be 95 to 98 per cent accurate done by the capable radiologist using his favorite dye and special techniques The test is dependable for evaluating the function of the gallbladder and cystic duct providing (a) the oral dye is absorbed from the intestinal tract (b) there is adequate liver function to excrete the dye and (c) the ductal system or gallbladder is not obstructed or diseased The cholecystogram should be able to identify

1 gallbladder function
   a normal
   b poor (faint visualization)
   c non function (obstructed)
2 pre ence of calculi in gallbladder
   a positive (or negative) shadows
   b presence of layering
   c calculi may be fine grains small pellets large concretions round facetted single or multiple with variations in size density composition shape and arrangement
3 the state of the common bile duct (particularly with the u e of Cholografin)
   a in the normal indicating size location and content (fig 42)
   b in the cholecystectomized individual who has adequate liver function (fig 43),
4 anatomic configuration of the biliary tract
   a locations of ducts and gallbladder in general and in relation to other structures in multiple views in the erect and recumbent positions
   b congenital inflammatory neoplastic or adventitious (including trauma) changes
5 other conditions
   a degrees of failure of the gallbladder to empty
   b state of liver function
   c tumor in adjacent to or near the biliary tract
   d complicating renal hepatic soft tissue diaphragmatic pulmonary or osseous disease

In most studies the failure of the gallbladder to visualize indicates di ease within the gallbladder—including biliary tract lithiasis stasis within the

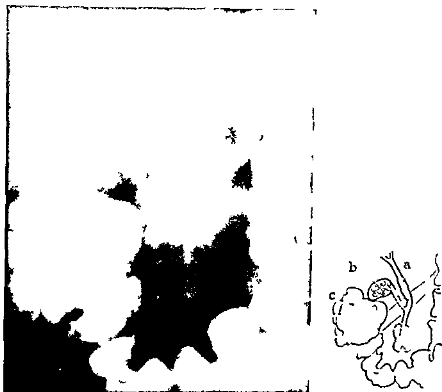


Fig 42A

Fig 42 The Common Bile Duct Visualized in the Intact Patient

A After failure of visualization by oral cholangiography Cholografin® was given intravenously The common bile duct (a) and at least nine calculi in the gall bladder (b) are seen Barium in the colon (c) is residual from previous examinations

B In the presence of anorexia nausea and obstructive jaundice intravenous Cholografin® may visualize the biliary tract as it did in this case when the jaundice was due to cholangiolitis (chlorpromazine cf Figs 38 and 91) Planigrams at several levels reveal a normal biliary tract no calculi or other obstructive mechanisms were present The cystic duct in this case is implanted on the left (medial) aspect of the common bile duct (cf Fig 23B)

biliary tract and inflammatory, fibrotic or malignant disease of the bile ducts and gallbladder

However additional causes for faint or non visualization may occur (a) during the last trimester of pregnancy, (b) during lactation when the dye is excreted by the breasts, (c) in severe uncontrolled diabetes (d) with inability to ingest or to retain the dye vomiting and diarrhea (e) because of delay in gastric emptying due to pylorospasm or organic pyloric obstruction (f) by reason of parenchymatous or other severe liver disease (g) in presence of intra abdominal inflammation (h) disease of the small bowel (i) excessive emptying rate of the gallbladder as associated with hyperchlorhydria and (j) due to the effect of drugs

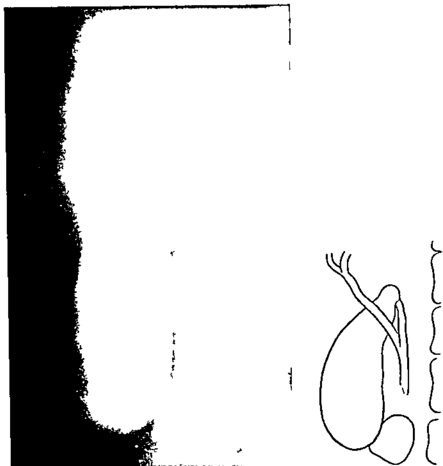


FIG 42B

Except in the use of Cholografin it is not expected that the common bile duct will be visualized in the course of the examination (figs 42 and 43) Using Telepaque visualization of the common duct has been reported to occur in up to 50 per cent with Priodax visualization occurs in less than 10 per cent of patients using tetraiodophenolphthalein visualization occurs in less than 0 1 per cent of patients The fact that the duct is seen during cholecystography is not abnormal Frequency in visualization of the common duct may depend on the intensity of search for it Planigraphic studies can be particularly valuable

D Operative Cholangiogram

Each patient at cholecystectomy is prepared and positioned for operative cholangiography Because of this and the increased exposure to the radi

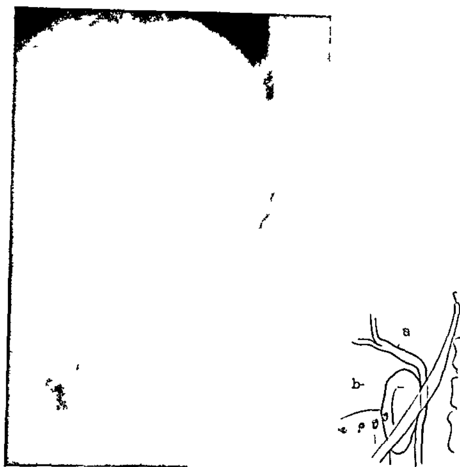


FIG 43 COMMON BILE DUCT VISUALIZATION IN THE POST CHOLECYSTECTOMY STATE (INTRAVENOUS TECHNIC)

Normal size position and contours seen in the extrahepatic ducts (a) The right kidney is visualized (b) (Re operation was avoided in this patient Relief from symptoms was obtained after psychotherapy)

ation operating room personnel must take precautions for themselves as well as for the patient It is preferred that the operating team be out of the room when x rays are done In the case of the surgeon and the anesthesiologist the use of specialized garments (lead glass aprons[1]) is indicated The surgeon's hands must be away from the direct radiation otherwise the duct should be injected before x rays are taken Special lead glass gloves[1] are available It is assumed that all electrical equipment is grounded that possible patient sensitization to drug is avoided and that proper anesthesia is selected

Before anesthesia is started the left side of the patient is raised 10 to 15 degrees by placing a folded sheet, blanket or sandbags below the left lower

[1] Owens Corning Glass Co (Bar Ray Co)

chest and pelvis The purpose of this is to rotate the vertebral column to the left and away from the distal end of the common bile duct A plywood tunnel (14 by 17 inches, 36 by 43 cm) which contains the cassette with a portable Bucky grid is positioned so that the bottom of the plate will be on a level with the crest of the ilium The cassette is centered at the approximate location of the cystic duct It is judicious to make a test exposure to insure proper operation of radiographic equipment

When the common bile duct has been opened the operating surgeon may do a cholangiogram through a catheter or 'T tube before concluding the operation (figs 44 and 45) It is also possible to visualize the common bile duct radiographically without incising the duct

1 A catheter tube or large needle may be placed into the gallbladder and dye injected through it to visualize the ducts (fig 46)
2 Intubation of the cystic duct by ureteral catheter before cholecystectomy or by use of the blunt needle through the cystic duct may visualize the duct system (fig 47)
3 If the cystic duct has been cut it is possible to intubate the stump of of the cystic duct by urethral or ureteral catheter (fig 48)
4 With the cystic duct ligated needle injection of the common bile duct may be done (fig 49)

In addition determination of intracholedochal pressure changes may be done by visual manometric and/or radiologic methods through the common duct or through (cholecystocholangiogram) the gallbladder (chapter 12)

A catheter within the bile duct (or gallbladder) is attached by means of additional tubing to a syringe which is filled with solution All air bubbles are eliminated from the system The significant factors in this procedure are (a) the absence of leaks around the point of insertion (b) the absence of air bubbles in the system (c) the removal of all instruments sponges and wound clips (d) the proper positioning of the patient the x ray cassette and the tube and (e) the proper protection of the personnel from exposure to radiation

At a selected time in the operative procedure either 35 or 70 per cent warmed Diodrast solution is prepared Previous irrigation to test the patency of the tube the emptying of the duct and the presence of leaks will have also given an estimate as to the volume of the bile ducts The normal duct will hold from 8 to 12 cc A large duct may hold from 20 to 30 cc The normal duct will empty from 1 to 3 cc per minute Flow through the wide open papilla may occur as rapidly as the duct is filled This information is required for the individual case

The operative field is covered with a sterile sheet or towel the location of the common duct identified The radiologist s equipment is placed in

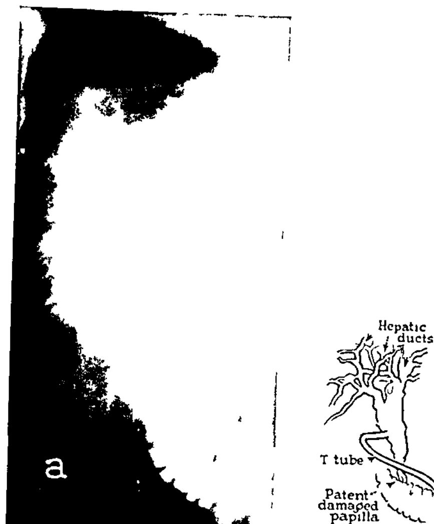


Fig 44A

Fig 44 Operative Cholangiogram

A Wide dilatation of the bile duct is found The patient had longstanding chronic calculous cholecystitis and choledocholithiasis Several duct calculi were removed just prior to this cholangiogram

B Trauma to termination of bile duct permits radio opaque media to enter the pancreatic duct for a short distance Cystic duct is filled up to its ligature (cf Chapter 11) Cholecystectomy done for lithiasis

position The anesthesiologist is forewarned and his cooperation obtained The anesthesiologist announces that he is ready to produce an apnea (or to instruct the patient to hold his breath if he is awake) If all the personnel are properly protected and away from the field injection is begun 8 to 10 cc are injected into the duct The patient is rendered apneic the radio-

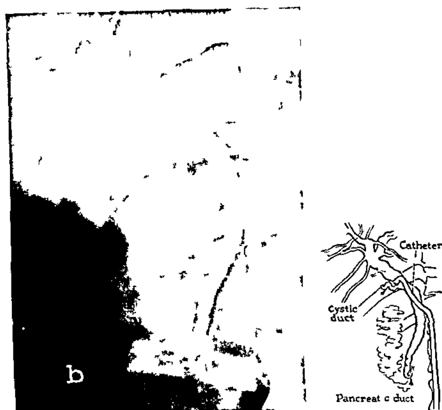


Fig 44B

graph is taken The injection is continued until from 15 to 30 cc of opaque media has been injected The cassette is changed and after two minutes another radiograph is taken

The cholangiogram can be taken by portable x ray machine capable of 60 milliamperes at 70 kilovolts or by a similar unit The radiographic unit may be a permanent part of the operating table If a Polaroid radiograph unit has a Bucky attachment it can be used successfully

The common bile duct on direct cholangiogram will present a shadow which depends upon the flow of contrast medium into the duodenum the presence of extrinsic deformity along the course of the common or hepatic duct and the existence of addition or subtraction filling defects in the course of the hepatic common bile or other ducts It is particularly important to be able to visualize all three major branches of the hepatic ducts Reflux from the common bile duct to the pancreatic duct may be normal in 5 to 15 per cent

The cholangiogram may have technical errors as follows (a) air bubbles,



Fig. 45 Operative Cholangiogram Indicating Correctable Errors

These include (a) tenting of duct at site of intubation (b) abnormal pressure of T limbs against duct wall (c) inadequate flow into duodenum and (d) residual choledocholithiasis

(b) too little contrast medium, (c) too much contrast medium, (d) extravasation of dye through adjacent periductal tissues (e) spasm of the termination of the duct due to the cold solution or other factors, (f) defects due to positioning wherein the common duct is covered by bowel or obscured by superimposed bone shadows (g) the end of the tube in the duct presenting a false positive shadow (h) stones in the (remaining) gallbladder overlying the hepatic radicles or the common duct

Air bubbles are fairly common Air bubbles change in shape and size and shift about Mucous floccules are often seen in the intrahepatic branches of the biliary tract The filling defects they cause are usually not of the roundish shape presented by stones but more irregular and often elongated Blood clots probably never occur in the bile ducts before instrumental ex



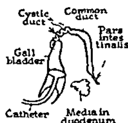


Fig 46 Cholecystocholangiogram

Normal operative cholecystocholangiogram except that hepatic ducts are not visualized This was done at the conclusion of nephropexy to exclude biliary tract disease following removal of benign lipoma of gallbladder fundus



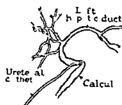
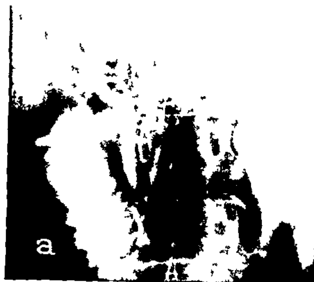


Fig 47 Operative Cholangiogram Using Ureteral Catheter

Ureteral catheter inserted through cystic duct Cholangiogram reveals normal flow through papilla into duodenum hepatic ducts are not abnormal two calculi in the common duct



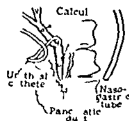




Fig. 48 Operative Cholangiogram

A Using urethral catheter inserted through stump of cystic duct Cholangiogram reveals choledocholithiasis distal segment of pancreatic duct is filled normal flow into duodenum (For an improved radiograph this patient should have been rotated ten degrees laterally to the right posterior oblique projection)

B Using "T" tube in the common bile duct Radio opaque medium rapidly enters duodenum Air bubbles are present in "T" tube and in the common bile duct



FIG 49 OPERATIVE CHOLANGIOGRAM USING NEEDLE PUNCTURE

Diodrast[R] injected through needle puncture of common bile duct Hepatic ducts (a) are incompletely visualized Radio opaque media has extravasated (b) from injection site Flow of media into duodenum is unimpeded (c) Although papilla is not visualized the bile duct position course and size are within normal limits

ploration A tumor in one of the primary hepatic ducts produces imperfect filling of the hepatic tree Dilatation of the primary hepatic ducts may be due to any constricting or expansive growth or obstructing stone In patients with cholangitis the roentgen picture of the hepatic branches shows irregular filling and flocculent shadows The ducts are ill defined and may have varying sizes

The normal space between the branches of two hepatic ducts may sometimes give the impression of a filling defect Several cholangiograms at different angles will generally clear up any doubt

Incomplete filling of an hepatic branch may be an indirect sign of disease Incomplete filling of the entire hepatic tree is nearly always due to mechanical obstruction of the duct Sometimes the tube may completely or partly obstruct one of the hepatic branches Attention should therefore be given to the manner in which a branch is filled

At surgery it is occasionally useful to cannulate the pancreatic duct or the hepatic ducts Radiographs taken after injection of dye according to the general technique described above are valuable

Certain ill effects have been noted following operative cholangiography These may be due to the chemical or toxic effects of the dye or to the dis

semination of infection Martensson has reported at least two deaths associated with *colon bacillus cholangitis following this procedure* *In most* cases, any abnormal reaction due to the procedure exists as a transient episode of pain and fever

F Postoperative Cholangiogram

The postoperative cholangiogram may be done *through* a tube in the gallbladder (cholecystocholangiogram), in a liver duct or in the common bile or common hepatic duct (It may be done after catheterization of an external bile fistula) The usual postoperative cholangiogram is done five days or later after operation in order to permit a natural seal to occur at the site of intubation and for adequate normal bile flow to be resumed

The day before cholangiography is scheduled the surgeon should evaluate the common duct clinically The duct volume and pressure can be



Fig 50 Normal Postoperative Cholangiogram

a right anterior hepatic duct b right posterior hepatic duct c left hepatic duct d common hepatic duct e common bile duct f *pars intestinalis* of the bile duct g contrast media in the duodenum



FIG 51 RECURRENT CHOLEDOCHOLITHIASIS

After choledocholithotomy 16 months previously postoperative cholangiogram was normal. Second choledocholithotomy was done 1 month previously. Cholangiogram 2 weeks after the second choledocholithotomy reveals residual choledocholithiasis. (At third operation *papillotomy done and Cattell type T tube was inserted*; tube removed after 1 year. Patient has been well for past 2 years.)

A Serial cholangiogram in Trendelenburg position reveals calculi against end of tube. Leathery deformity at duct termination indicative of previous calculous trauma.

B Cholangiogram in erect position permits calculi to gravitate towards distal termination of the duct (Bore idis)

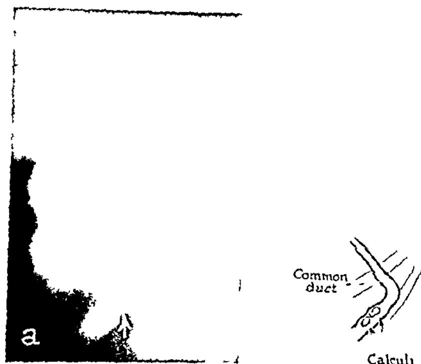


FIG 52 RESIDUAL CHOLEDOCHOLITHIASIS

Cholecystectomy for lithiasis was followed after 3 months by severe colicky pain without jaundice

A Three years later Cholografin® study indicated residual choledocholithiasis

B Operative cholangiogram (after removal of two stones from the common duct) *indicates a third calculus* This was *removed* and transduodenal papillotomy was done No medium enters the duodenum

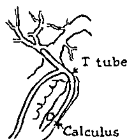
C Postoperative cholangiogram after ten days reveals normal duct A long limbed "T" tube passes through the papilla

measured and these *findings* given to the radiologist for his guidance The clinical rate of emptying is most important to the value of the study (chapter 15)

The patient is usually placed in a right posterior oblique and recumbent position It is feasible that specific apparatus (Bornholdt) may be attached to permit mobility of the patient, stability of the tube and improvement in technique Either 35 or 70 per cent Diodrast is used and the tube system *is prepared to be free of air No injection nor aspiration should be done from* the system in the 24 hours preceding the cholangiogram It is preferred that the patient not eat during the preceding four to six hours although this is not absolutely essential A small dose of phenobarbital (gr $\frac{3}{4}$ $\frac{1}{2}$) may be given to the patient Severe and strong catharsis and other medications should be avoided

The injection is done in stages a small amount of medium is injected under fluoroscopic control and a *preliminary large film taken* An addi







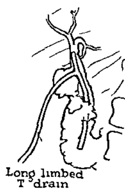


FIG 52

tional 1 to 3 cc. of medium is injected and repeated serographic films of the distal duct and duodenum are exposed. A small amount of Diodrast is *instilled immediately* before each exposure (fig. 51). Large films are taken to demonstrate the entire hepatic ductal system in recumbent and Trendelenburg position. The patient is then raised to the erect position and the studies are repeated.

In the cholecystocholangiogram, the initial injection of medium should be at least 30 cc.

Intervals between fluoroscopic and radiographic studies are essential to evaluate the function and emptying capacity of the duct system. Administration of morphine may prolong retention of medium when flow is too rapid. *Papaverine may permit increase in flow. Other methods are useful.*

1. The Normal Cholangiogram

The origin of the common bile duct is usually in the right half of the body at the median sagittal plane at approximately the 12th thoracic



Fig. 53. Residual Choledocholithiasis

Postoperative cholangiogram shows choledocholithiasis (a). [illegible] therapy was utilized for 4 months. The common duct stone disappeared. Following removal of the tube the patient has been free from symptoms for over 7 years. (Radiograph courtesy of Ann. Surg.)

vertebral interspace (fig 50) The normal common bile duct will contain from 8 to 10 cc of fluid and will empty at a rate of 1 to 3 cc per minute Under fluoroscopic observation contrast media injected through the T tube normally will enter the common bile duct and thence the duodenum with delay or obstruction The duct is seen as a constant curve (double S shape) generally parallel to the vertebral column presenting several changes in direction At its termination the duct lumen decreases in diameter and presents a double concavity or scalloping which gives the lumen at the termination of the duct the appearance of a funnel It is rare to see the thin filamentous canal which represents the transpapillary common bile duct but it is not abnormal to do so Normally the duodenal canal fills rapidly above and below the terminal orifice of the common bile duct The last one half to three centimeters of the extra duodenal common bile duct may be straight and rigid (transpancreatic)

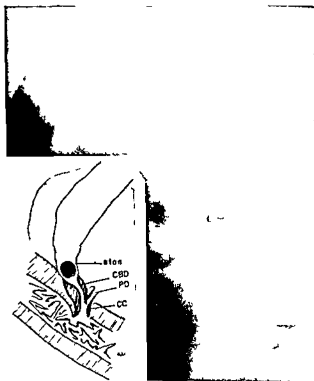


Fig 54 Residual Choledocholithiasis

Postoperative cholangiogram reveals residual stone impaction proximal to papilla and proximal to site of origin for reflux into pancreatic duct (Courtesy of *A M A Arch Surg*)

2 The Abnormal Cholangiogram

Abnormalities usually observed include choledocholithiasis, papillary spasm, duct stricture, tumor, intraductal reflux and periductal extravasation.

The most common abnormality is residual choledocholithiasis (figs 51, 52, 53 and 54). The calculus may be a positive or negative shadow, circular, crescentic or irregular in shape, but always constant. It is fixed in appearance but not in location. It is usually heavier than bile and seeks a dependent position. A stone may not be mobile if it is fixed in a diverticulum or is impacted against a mucosal fold. Impaction of calculi most frequently occurs at the termination of the extraduodenal common bile duct (fig 54).

The damaged duct has a greater capacity than normal in most cases. When contained volume is greater than 12 cc, the duct must be considered to be abnormal until disproven. However, if a calculus has recently been



Fig 55 Intraductal Reflux

Bile duct termination is normal. Pancreatic duct fills from the duodenum by reason of lower pressure gradient. Pancreatic duct fills partially (2) and empties (1) leaving small residual segment.



FIG 56 POSTOPERATIVE CHOLANGIOGRAM ABNORMAL FILLING OF PANCREATIC DUCT

Pancreatic duct fistula was produced at duodenotomy by use of probes Choledochoduodenostomy was required for stricture at papilla 8 months later Patient well since (3 years) (Radiograph courtesy of *Am J Gastroenterol*)



FIG 57 POSTOPERATIVE CHOLANGIOGRAM

Dyskinesia at papilla associated with deformity of duct termination and dilatation of common bile duct Tumor suspected but micro copy was benign (Radiograph courtesy of *A M A Arch Surg*)

removed from that duct, its larger volume is readily explained This duct usually empties rapidly

Flow of contrast medium into the pancreatic duct from the bile duct may be abnormal Interductal reflux is normally seen in approximately 10 per cent of patients after dye from the common bile duct has entered the duodenum (fig 55)

An additional 5 to 10 per cent of patients will demonstrate an abnormal type of interductal reflux The contrast medium will enter the pancreatic duct from the common bile duct *prior to the* appearance of dye in the duodenum This is usually associated with trauma, *often* due to calculous disease or surgical manipulation at the papilla (fig 56) In such patients repeated cholangiograms often reveal a normal duct later

Spasm present at the termination of the duct will be demonstrated by resistance to flow and decreased rate, however duct volume is not in



Fig 58 Postoperative Cholecystocholangiogram

Arrow at distal end of common bile duct indicates (unsuspected) diverticulum at the papilla Choledocholithiasis is not proven

creased Injection of the duct may be painful but the level of perfusion pain is usually less than 20 cm of water

If the capacity of the duct is greater than 15 cc, and the pain threshold over 25 cm of water, it is likely that "dyskinesia" has replaced spasm and it may be concluded that biliary tract dystonia is present (fig 57)

In one third of patients the last 2 or 3 cm of the extraduodenal common bile duct is totally surrounded by pancreas This may be recognized when the transpancreatic segment is straight, rigid and smaller than the other parts of the common bile duct Diverticula are often seen in patients with



FIG 59 POSTOPERATIVE CHOLANGIOGRAM

Pancreatic duct reflux from bile duct following trauma associated with choledocholithiasis and previous choledochotomy Marked deformity of the common hepatic duct is due to extrinsic pressure from the left by a pancreatic pseudo cyst Interductal reflux occurs in presence of damage at papilla despite free flow of media into duodenum

pancreatitis (fig 58) Pancreatitis can occlude the common bile duct and deformity due to pressure is often seen in the distal duct In other cases, pressure from an extrinsic cyst can cause marked sweeping deformity in the course of the common or hepatic ducts, often associated with some diminution in caliber (fig 59)

Stricture of the common bile duct offers complete or total resistance to bile flow More than 20 cc of fluid may be injected into the duct but none will enter the duodenum The hepatic radicles are usually dilated and frequently there is associated extravasation of dye around the tube The diameter of the duct proximal to a stricture is usually greater than 18 mm The distal segment of the duct is blunted deformed and distorted (fig 60)

Differentiation of stricture can, in some cases be made radiologically Stricture at the papilla can be malignant, traumatic or congenital These are not common, diagnosis is often by inference and by failure of dye to appear in the duodenum In the extraduodenal common bile duct an inflammatory stricture may be identified by reason of the long narrow ir



Fig 60 Stricture of the Common Bile Duct

Postoperative cholangiogram reveals a blunted occluded distal common bile duct All segments of the extrahepatic biliary tract are dilated This first stage choledochostomy was followed by choledochojejunostomy



FIG 61 INFLAMMATORY STRICTURE OF THE COMMON BILE DUCT

Cholangiogram following cholecystectomy and choledochotomy for lithiasis The common bile duct is of very small caliber The common hepatic duct is dilated Intra hepatic ducts are normal Choledochostomy was followed later by choledochoduo denostomy (*Radiograph courtesy of Am J Gastroenterol*)

regularity of a semirigid duct (fig 61) Traumatic stricture is very short The dilatation proximal to traumatic stricture is often massive that as sociated with inflammation may be minimal Neoplasm of the duct or periductal tissues which produces incomplete stricture may be identified (in the rare case) by irregular pattern of addition and subtraction defects

Dilatation is always present in the ducts on the hepatic side of a stric ture If any area of the duct particularly at the papilla is suspected of being the site for stricture proximal dilatation should be present

The serial cholangiogram is particularly valuable for studying the papilla and duct (figs 62a and 62b) A long limbed (Cattell) tube does not prohibit cholangiographic study it is best done in the Trendelenberg position fig(63)

3 Summary of Radiographic Anatomy

The common bile duct as seen by the radiologist is a constantly curving tube often as a reverse "S," and occasionally with some acute angulation which is composed of intrahepatic, extraduodenal and transduodenal segments The most distal portion of the extraduodenal common bile duct is

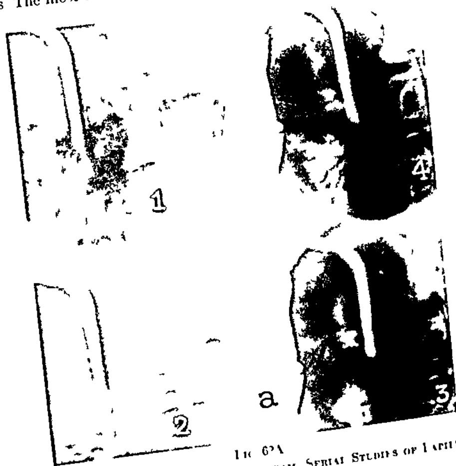


Fig 62A

Fig 62 Postoperative Cholangiogram Serial Studies of Papilla

Courtesy of J Gershon Cohen and A Boreadis

A Normal papilla constant changes in the papilla occur as the common bile duct empties radio opaque media through the papilla into the duodenum

B Damaged papilla examination of patient in the Trendelenburg position reveals a dilated bile duct which contains ever the ... adjacent to the distal horizontal limb of the T tube The papilla is deformed the duct termination is irregular and feathery The entrance of the media into the duodenum is irregular and sluggish

C Effect on papilla and the bile duct of prolonged adequate choledochotomy is seen on comparison of cholangiograms taken on December 19 195 (upper series) and those on January 5 1953 (lower series) Bile duct diameter decreased The papilla is restored to normal appearance

transpancreatic in about one third of cases and can be identified as such when the duct is straight, semirigid and occasionally smaller than the other portions of the extrahepatic bile ducts.

The diameter of the normal extraduodenal common bile duct on the cholangiogram averages 7 mm. The average diameter of the orifice of the common bile duct within the duodenum on the x-ray plate is 1 to 2 mm. The abnormal common bile duct is one which is greater than 1 cm. in diameter and which contains defects or presents abnormalities in its course.

The normal transduodenal segment of the common bile duct on the x-ray has a funnel-like shape and a decreasing diameter. The termination of the common duct is a curving, filamentous canal which may normally be 0.5 to 1.5 cm. in length.

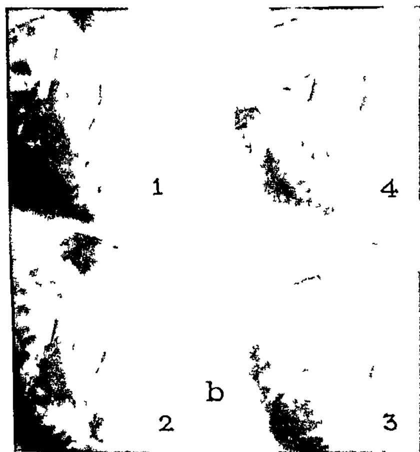


FIG. 62B

which are greater than 2 cm in diameter Workers in this field have reported 93 per cent accuracy

False positive reactions occur in both types of studies These may appear in patients with acute hepatitis and in portal cirrhosis with and without ascites In addition, an active gastric ulcer may pick up the isotope

Thorium dioxide has been used with limited application for identification of metastatic malignancy in the liver

At present, use of radioactive isotopes is limited in scope for clinical application to the liver and biliary tract

In the laboratory however, trace element radio-spectrographic methods are extremely valuable in evaluating biological potential Olson and his collaborators have found molybdenum zinc, iron chromium and cobalt in the liver These have been found in abnormal concentrations in carcinoma of the esophagus metastatic carcinoma to the liver, lymphatic leukemia and portal cirrhosis It would appear that these elements are linked to protein molecules as essential components in enzyme systems For example, zinc is part of the carbonic acid anhydrase and possibly of some peptonases Copper is present in tyrosinase and *molybdenum is* probably part of the xanthine oxidase Detection of these elements in *serum liver and bile will provide much information regarding* cellular and hepatic function

2 Visualization of the Hepatic Circulation

Intravascular catheterizations may be used for visualization of the hepatic circulation Diodrast injected into the spleen can fill the portal vein and its tributaries as a useful diagnostic tool Success in this method depends upon enlargement of the spleen and the accuracy of its location With the patient in the prone position, a 17 gauge needle is introduced into the left 9th or 10th interspace along the *posterior axillary line (Irvine)* The needle is directed upward medially and hugs *the internal surface of the rib cage to enter the spleen* A brisk flow of blood into the syringe emerges when the needle engages the spleen Twenty cc of Urokon (or Diodrast) is rapidly injected during two seconds When the injection is finished radiographic exposure is made If a rapid cassette changer is available exposures are taken as rapidly as possible starting after the first 5 cc have been introduced Lead glass lead rubber or other protective equipment for the hands is obligatory

At present aortography for visualization of the *celiac axis and hepatic* artery circulation is not practicable

On the other hand *hepatic venous outflow* can be *demonstrated* after catheterizing the right auricle through the superior vena cava *The catheter may be positioned fluoroscopically* in the hepatic veins and several roent

genograms are taken after the rapid injection of approximately 20 cc of contrast medium while the patient holds his breath in expiration

Chapter 8 was prepared in collaboration with Morton Hermel M D and after advice by Jack Gershon Cohen M D and Raymond Katzen, M D

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9

MEDICAL MANAGEMENT

A Management in the Absence of Complications

Biliary colic is a frightening emergency to the patient It should not be so to the physician There is no need to stampede into emergency surgery *for relief of the acute infection or obstruction to the gallbladder and bile ducts*

The patient who has a diseased gallbladder may have any of five situations which accompany severe biliary colic 1) pain, tenderness, fever *and/or a mass in the right upper quadrant, 2) nausea vomiting and/or* diarrhea accompanied by electrolyte imbalance, 3) jaundice and its sequelae *4) hemorrhagic phenomena secondary to liver insufficiency, 5)* other complications such as pancreatitis hepatic failure, renal insufficiency and coronary artery disease

Each patient with disease of the biliary ducts or gallbladder should be managed both individually and with due regard to a general plan

1 Determine the nature of the biliary tract disorder
2 Evaluate the general condition
3 Support the patient by parenteral feeding and antibiotic therapy
4 Confer with internist cardiologist gastroenterologist, clinical pathologist radiologist and anesthesiologist as indicated

Carefully planned management can decrease the postoperative morbidity in gallbladder disease At present there is a mortality rate of approximately 6 per cent whether the patient is hospitalized at a small general hospital in Wisconsin or Georgia or at University Centers in New York or Chicago This 6 per cent mortality rate is due in nearly 80 per cent to associated pulmonary infection *cardiac disease renal damage malignancy* and degenerative disease It is important for the doctor to realize that *death in the patient with gallbladder disease* is often not related to the biliary tract

When evaluation of the patient with biliary tract disease has been adequate and proper therapy provided preoperatively the mortality rate decreases to 1 8 to 3 2 per cent (Glenn et al)

1 Nature of the Biliary Tract Disease

Even typical clinical symptoms of biliary *colic must be confirmed* Positive radiographic finding of gall stones are usually sufficient for the diagno-

sis However, patients with symptomatic duodenal ulcer or pancreatitis may also have a calculus in the gallbladder Therefore repeated examination and evaluation of such patients may be necessary In the acute episode without pre existing positive evidence of gallbladder disease the patient is treated conservatively Adequate information can be obtained after acute symptoms have subsided

It is necessary to exclude disease of the stomach including hiatal hernia acute and chronic gastritis gastric ulcer or malignancy polyps and gastric mucosa prolapsing through the pylorus disease of the duodenum such as diverticula polyps and ulcer disease of the appendix disease of the cecum including cecal ulcer carcinoma and diverticula It is also necessary to exclude one of the many forms of pancreatitis In certain patients hepatitis is a frequent cause for pseudobiliary colic There have been patients with drug addiction tabes dorsalis herpes zoster or intercostal neuralgia whose symptoms closely resembled those of biliary colic Other factors associated with symptoms are those of diaphragmatic pleurisy pneumonia hyper sensitivity and cardiac insufficiency

No matter how closely a symptom picture may resemble that of biliary colic it is essential that clinical laboratory and x ray findings be in agreement to make the positive diagnosis of gallbladder disease

2 Evaluation of the Patient

The problems in biliary tract surgery frequently are not related entirely to the bile ducts Hypertension diabetes arteriosclerotic and other forms of heart disease peripheral vascular disease hepatic failure renal disease pneumonia and carcinoma account for a great portion of morbidity and mortality

Careful evaluation is needed in the patient who may have both coronary artery and biliary tract disease and symptoms due to either (Walters and Master)

The patient who has been under careful medical management and observation for some time may not require prolonged hospitalization for elective surgery However in the acute episode it is not fair to the patient or to the surgeon to perform biliary tract surgery until all factors regarding the patient s general condition have been evaluated and the patient given an opportunity to compensate for any deficiencies

3 Preoperative Preparation

Preoperative preparation is divided into three phases clinical laboratory and specific individual requirements

a Clinical

The patient's clinical history is carefully investigated In some older individuals certain episodes indicating a pre existing 'silent stroke' may be elicited In others angina or renal disease may be found Carcinoma of the gastrointestinal tract or elsewhere may exist Blood pressure pulse rate and weight changes may indicate a latent or burned out hyperthyroidism If indicated repeated electrocardiograms and other functional tests are accomplished to correlate the patient's clinical status

Physical findings are noted and rechecked as often as necessary These patients may be examined three or more times daily to determine whether intra abdominal complications directly related to the biliary tract are present Emergency cholecystostomy is occasionally required

b Laboratory

Laboratory examinations are accomplished as follows Complete blood count is done and, if indicated, platelets bleeding time and clotting time are checked In all patients with suspected biliary disease the urine is examined, particularly for the presence of bile and urobilinogen Urine is examined daily in patients with jaundice, to determine whether there is change in the urobilinogen level Sedimentation rate, blood urea nitrogen blood sugar prothrombin time and serology are done in all cases

The results of these tests may indicate further examinations If there be any abnormalities, then additional studies are done 1) total proteins including albumin globulin ratio, 2) bromsulfathalein excretion 3) Van den Bergh, 4) liver flocculation studies

When vomiting or diarrhea have been present it is essential to determine the plasma chlorides and the blood bicarbonate (CO_2-combining power) In seriously ill patients with complicating disease, it may be necessary to check blood volume and total base However the patient who requires these latter studies is usually so ill that surgery is to be deferred

c Individual Studies

Additional studies are accomplished as indicated in the individual patient These are done in cooperation with the x ray department cardiologist gastroenterologist, anesthesiologist and the patient's physician Sigmoidoscopy may be indicated If sufficient suspicion exists the cholecystogram can be done at the same time as a barium enema If there be additional doubt regarding status of the gastrointestinal tract, the esophagus stomach duodenum and small bowel are studied radiographically

Consultation may be required for the patient's general condition This is essential in, for example, the patient with both hiatal hernia and cholelithiasis the patient who has both duodenal ulcer and cholelithiasis the

patient with angina pectoris and obstructive jaundice and the patient who has obvious severe disease of the biliary tract together with recurring episodes of cardiac failure

The individual with gallbladder disease in the older age group has been exposed to so many possibilities for degenerative, inflammatory or other ailments that he must be studied carefully under all circumstances

Inhalation anesthesia is preferred (endo-tracheal) with relaxation obtained by use of curare like agents There are certain situations which are best managed by spinal or local anesthesia or by other inhalation anesthesia agents (chapter 10)

All studies completed and all difficulties corrected the patient s attitude and frame of mind should be optimal A broad spectrum antibiotic such as Terramycin or Aureomycin (one or more grams daily) is given orally for three to four days preoperatively The parenteral use of Streptomycin (one gram) and Penicillin (600 000 units) daily for two or three days preoperatively is also effective

The individual who has been vomiting may require nasogastric suction The gastric tube is unnecessary in most patients In most cases intake is restricted to water for ten to twelve hours preoperatively and absolutely for four to six hours Preoperatively an enema is given only if necessary

4 Diet

Whether hospitalized or under home care much benefit is derived from adequate diet These patients are given intravenous feeding only if anorexia nausea or vomiting is present When appetite returns (even though the patient is afraid to eat) liquids except milk are given followed by jello crackers and chicken Gradually high caloric foods and milk are added A full diet may be taken except for chocolate cabbage spices and fried foods The patient should be not only well hydrated but also adequately nourished

A sample menu for a patient with chronic cholecystitis is as follows

Breakfast

Orange juice chilled
Oatmeal or dry cereals (no bran) with milk and sugar
Hardboiled egg (1)
Crisp bacon
Toast with taste of butter
Milk coffee or tea with milk and sugar

Dinner

Grapefruit
Consomme spinach (without cream)
Roast lamb with mint sauce
Baked potato with very little butter

Carrots and celery
Sliced tomatoes with vinegar and sugar
Toast white or rye bread
Soft custard with fruit sauce jello
Milk coffee or tea with milk and sugar

Supper

Clear soup with rice
Boiled chicken
Hard rolls or Melba toast
Stewed pears jello
Glass of milk coffee or tea with milk and sugar

5 Summary

The preoperative care of the patient with uncomplicated gallbladder disease includes 1) individual management, 2) identification of cause for disease 3) recognition and treatment of associated factors in the liver *intestine cardiovascular renal peripheral vascular and central nervous* systems, 4) maintenance of normal function in the presence of degenerative phenomena such as arteriosclerosis, 5) prevention of activation of *bronchosinusitis allergies or renal insufficiency, and 6) maintenance of* adequate nutrition

The patient with acute biliary colic should be subject to surgery whenever his general condition permits *it but not before* positive diagnosis regarding the bile ducts and the patient's organic functions have been clarified

The indication for surgical procedure is the presence of symptomatic biliary tract disease Election of time and procedure indicated is always individualized

B Management in the Presence of Complications

The patient with biliary tract disease may *have complicating phenomena* such as severe pain, jaundice pancreatitis or infection

1 Pain

Pain may be primarily of biliary tract origin or may be referred from extrabiliary sources In the biliary tract the following are considered (a) *impaction of stones or debris in the cystic* duct or at the papilla of Vater (b) hydrops or empyema mural infection or abscess in the gallbladder cholangitis or pericholedochitis or *inflammation of the* sphincter of Oddi (c) (imminent) perforation of the gallbladder or ducts (d) pancreatitis (e) leakage of bile or and pancreatic ferments into the retroperitoneal or peritoneal tissues *(f) pressure phenomena* secondary to spasm provoked

by the abnormal irritation and (g) reflex pylorospasm secondary to abnormalities within the biliary ducts

Another source for biliary tract pain is extrabiliary, i e, of cardiac, pulmonary, neurogenic, gastric, pancreatic or renal origin

Nitrites, papaverine and atropine in large enough doses may frequently overcome spasm If pain is not thereby relieved, then morphine, Demerol, Pantopon or Dilaudid in adequate dosage is used It is noted that most pain due to the biliary ducts is organic rather than due to spasm If neither the antispasmodics nor the narcotics afford sufficient relief, it is probable that intravenous novocain or paravertebral block (including splanchnic block) may be indicated A simple and harmless way to obtain quicker relief in cases of severe stone colic is 0 1 per cent novocain slowly injected intravenously Pain subsides within ten minutes in over half the cases A transient hypotension appears in 38 per cent of cases A combination of novocain with papaverine, magnesium sulfate or phenobarbital alleviates distress in 80 per cent of the cases

2 **Infection**

Bacterial or viral infection may originate in the biliary tract and may secondarily involve the appendix, pancreas, colon and liver In addition there can be coincident or primary inflammation in these organs

Gallstones which impact in the cystic duct or which erode portions of the terminal segment of the extraduodenal common bile duct may inaugurate inflammation and abscess Hydrops of the gallbladder may become an empyema The simple common duct obstruction may become severe cholangitis or pericholedochitis

Infection in the biliary duct is indicated by (a) fever (which may rise to 102°) (b) a rapid sedimentation rate (Behrend) (c) a leukocytosis between 15 000 and 25 000 white blood cells with a polymorphonuclear increase and a shift to the left

In the presence of infection it is advisable to administer antibiotics During the early phases when there is an inability to retain fluids, penicillin and streptomycin (intramuscular) or Aureomycin or Terramycin (intravenously) are indicated Just so soon as oral intake is re established Terramycin or Aureomycin may be given orally It is not wise to persist indefinitely in antibiotic therapy

3 **Electrolyte Loss**

The patient with biliary tract colic pain or other disorders frequently has electrolyte imbalance In the handling of the non febrile patient who has no abnormal loss of electrolytes 1500 cc of dextrose and water and 500 cc of saline solution is adequate for increased sodium and chloride re

quirements The daily administration of 40 m Eq of potassium should satisfy the potassium requirements

In the seriously ill patient, the water requirements are estimated by totalling the known water loss and adding to this sum the estimated value for loss through the skin and lungs including losses by excessive perspiration, hyperventilation or fever The amount and type of electrolytes to be administered will depend entirely on the actual laboratory determination of these losses for the preceding 24 hour period

If prior to the administration of replacement solutions clinical data are not available as to the fluid and electrolyte losses one can use figure such as those given by Butler He reports that in a patient who has lost 10 per cent of his body weight the daily losses per kg of body weight are water 100 cc, sodium 7 m Eq, chloride 6 m Eq, potassium 7 m Eq A diabetic who has been without insulin for three to four days and has been vomiting for one day loses water at the rate of 100 cc per kg per day sodium at the rate of 5 m Eq per kg per day, chloride, 4 m Eq per kg per day and potassium, 6 m Eq per kg per day

In using these or comparable data for estimating needs, one should keep in mind a marked variation of body water content and distribution with age sex and weight The lean body mass of the average 70 kg male is much greater than that of the average 57 kg female due to the greater amount of fat in the female This fact is of importance since the total body water of the average male is 43 kg, representing 73 per cent of the lean body mass and the value of total body water in the female is only 29 kg although it represents the same percentage of water in relationship to the lean body mass Similarly the amount of intercellular water is 31 5 kg in the male as contrasted to 20 kg in the female Hence, if an average female and an average male lose a comparable amount of fluid through whatever body route on average, there is in the female a far greater loss of total body water and eventually of intercellular and interstitial water A far greater degree of hemoconcentration results than in the male so that more significant changes will be found Similarly the older patient requires much modification of so-called 'routine therapy

Hypochloremic acidosis may occur if there has been constant recurrent vomiting or if nasogastric suction has been required The major deficit here is in chloride and fluid This should be compensated

Protein hydrolysate or blood transfusions are required in the patient with malnutrition The individual who has a chronic biliary tract disease frequently refuses a diet because of recurrent anorexia and vomiting which have been produced by food Under these conditions it is essential to rebuild protein and glycogen levels

When symptoms are acute there may be an associated intestinal ob

gus (without dressing) endive string beans wax beans beet greens stewed celery mushrooms okra boiled squash stewed or canned tomatoes turnips vegetable marrow carrots beets boiled potato (no butter)

Bread As at breakfast

Desserts Apple sauce blanc mange (without egg) brown betty stewed or canned apricots cherries pears plums (water packed) custard (without egg yolk) fruit souffle (egg white only) gelatin jello junket puree of prunes Very ripe raw fruits as apricots peaches or pears without skins or seeds

Beverages One glass of milk or buttermilk coffee or tea

b *Low Cholesterol, Low Fat High Caloric Diet (This diet supplies approximately 350 gm carbohydrate 100 gm protein 170 gm fat and 3500 calories)*

Breakfast

Fruit and fruit juices Apple sauce baked apple sliced banana or stewed prunes with milk and sugar Orange grapefruit pineapple or prune juice sweetened if desired

Cereals Dry cereal (except bran) with milk and one teaspoonful of sugar

Bread Two slices of white bread corn bread or two soft rolls preferably toasted with one square of butter

Waffles or hot cakes May be eaten two or three times a week with syrup or honey

Jellies Apple currant or grape jelly apricot plum prune or strawberry jam honey or marmalade with breads

Egg One hardboiled or poached

Beverages Coffee (or substitutes) tea with one tablespoonful of cream and two teaspoonfuls of sugar homogenized milk

Luncheon and Dinner

Juices Tomato or vegetable juice

Soups Asparagus cauliflower celery chicken corn mushroom pea potato spinach or tomato made with milk (no cream) Consomme with barley noodle macaroni okra rice spaghetti or vermicelli

Meats (Meat or fish once daily) No fats Chicken roasted boiled or broiled Roast turkey Beef roast or broiled Steaks broiled tenderloin sirloin or round Lamb chops or roast leg of lamb Mutton broiled or chops Roast leg of veal

Or fish (Baked boiled or broiled) Bass blackfish bluefish cod flounder haddock halibut steak perch weakfish whitefish red grouper pollock cooked salmon Can be served with lemon juice

Vegetables Potatoes (sweet or white) boiled mashed or riced with half a portion of butter or macaroni spaghetti or rice with cheese mushroom or tomato sauce With two portions of colored vegetables as asparagus beets cauliflower string bean wax bean baked beans lima beans (young) green peas lentils mushroom okra boiled squash turnip stewed tomatoes vegetable marrow

Desserts Apple sauce apple snow baked apple (with sugar no cream) fruits stewed or canned as apricots peaches pears plums Banana fresh or baked Chocolate occasionally Cake sponge plain pound or cup (without frosting or chocolate) Puddings tapioca rice bread corn starch Junket Jello Ices sherbet or water ice Fresh fruits may be taken in season without skins or seeds if very ripe

Additional feedings at 10 a m 3 p m and 10 p m Malted milk Ovaltine Cocoa

malt, or milk to which one tablespoonful of corn syrup or cerelose has been added With toast, breadsticks or crackers Jelly, jam or honey if desired Additional protein supplements as Meritene, Levugen, meat extracts and skim milk powder are valuable

Avoid Fats, fried foods, pork, bacon, thickened gravies Inner organs as brains, kidneys and sweetbreads Olives, olive oil, salad dressings Pickled and smoked foods All rich and highly seasoned foods, condiments, salt and pepper at table Nuts, dates and figs Rough foods as cabbage, corn, Brussels sprouts Whole wheat and bran products Pies, pastries, chocolate Alcohol

5 Hepatic Insufficiency and Jaundice

The patient with biliary tract disease may have intermittent jaundice The degree of expansion of the bile ducts (pre-existing infections may prohibit dilatation of the ducts) and the quality of liver function are determining factors Jaundice may be complicated by hemorrhage, weakness and other signs of hepatic insufficiency

Obstructive jaundice is to be relieved (surgically) just as soon as the patient is in proper chemical and physiological balance It is not necessary to wait until the patient's jaundice has cleared This is not feasible in many cases Persistent jaundice will cause undue suffering as well as excessive damage to the hepatic parenchyma (chapter 4)

6 Other Complicating Factors

a *Pancreatitis*

Surgery is usually withheld in pancreatitis except for its complications The patient with biliary tract disease who has pancreatitis should be treated conservatively for a sufficient period of time (chapter 5)

b *Systemic Disease*

Great emphasis is placed upon treatment of cardiac irregularities, abnormalities and insufficiencies Proper hygiene of the mouth and oral pharynx should not be overlooked Bronchosinusitis or other chronic upper respiratory infections may be troublesome if prophylactic measures have not been instituted

The individual's gastrointestinal intake is stimulated at times to supernormal levels The patient with biliary tract disease who also has duodenal ulcer, hiatal hernia or gastritis requires aid to improve the oral intake and utilization of food Patients who have biliary tract disease may have either chronic constipation or intermittent diarrhea Special treatment may be required to treat existing hemorrhoids, fissure in ano and other similar rectal disease It is preferred that mild (saline) catharsis be used in place of repeated enemas

It is preferred that bland diets consisting of high carbohydrate and high protein feedings be utilized instead of rich cream feedings The diet should

avoid cabbage fish, butter, fried foods and spices Adequate fluids fruit juices and hard candies should be taken Chocolates and other candies which contain oils are avoided It may be wise to supplement the diet with protein hydrolysates such as Nutramigen Amigens, Levugen, Essenamines and other specially prepared food concentrates Vitamins B C and K are added

c Patient's Cooperation

The patient's cooperation is of utmost importance Mental stability and full confidence have to be obtained by understanding each individual's problems The dull miserable, apathetic and melancholy attitude which is frequently present is often a part of the biliary duct disease The patient are advised that biliary tract surgery has a lower mortality than its complications and that they *are to be much improved as a result of biliary* tract surgery This is discussed with members *of the family as well as with the patient so that a proper optimistic attitude may be obtained*

7 Summary

Management of disorders in the bile ducts when complicating disease is present requires 1) comfortable bile flow, 2) proper antibiotics, 3) adequate nutrition 4) that all systems in the body—cardiovascular peripheral vascular, cardiovascular renal pulmonary, upper respiratory and gastrointestinal—be given *sufficient therapy so that they are normal*

The assistance here of a qualified internist and proper specialists may be necessary

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10

ANESTHESIA FOR BILIARY TRACT SURGERY

LILLIAN E. FREDRICKS, M.D.[1]

A. Introduction

The progress of surgery in the last decade has been aided greatly by the advancements in anesthesiology. Teamwork between surgeons and anesthesiologists permits surgery for poor risk patients and those heretofore considered as technically inoperable.

Selection of the proper type of anesthesia depends upon the patient's history, physical findings and pertinent laboratory studies. The anesthesiologist should visit the patient before operation to establish rapport and to evaluate the patient's clinical status. Heart and lungs, blood pressure and pulse are examined and a history of previous anesthesia experience is taken to supplement the findings from the patient's chart.

It has become of importance to question patients regarding therapy with ACTH or Cortisone within the preceding few months. These drugs are used for treatment of numerous diseases and it is recognized that with prolonged use they suppress adrenal activity. The failure to recognize this may provoke sudden collapse of the patient who is exposed to additional stress such as anesthesia and/or surgery. In order to prevent this, Cortisone *is given in large doses (75–100 mgm.) the night before and the morning of* operation. Cortisone is continued postoperatively in diminishing amounts as individually indicated.

B. Pre-anesthetic Medication

The choice of the anesthetic agent, the method of administration and the premedication depend upon the type of operation contemplated and *upon the physical status of the individual patient. In biliary tract surgery* particularly, selection of the pre-anesthetic medication is of great importance. Biliary colic may be precipitated by morphine used for premedication. Such patients may have severe abdominal pain or nausea and vomiting associated with varying degrees of circulatory collapse within one half

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hour after the administration of the drug. Morphine primarily increases the tone of smooth muscle and to some extent the force of contraction (Salter). Pain may result due to this increase in muscle tone and the closing of the sphincter of Oddi. *Numerous investigations have been carried* out to show the influence of various drugs on the pressure in the biliary ducts. Among others, Curreri and Gale have shown that analgesics as a *group are powerful stimulators of the sphincter of Oddi.* This includes morphine, codeine and Dolophine. Demerol has variable effects but it is *less spasmogenic to the sphincter of Oddi than is morphine.* Atropine does not alter the pressure in the ducts. Because of these considerations we do not use morphine for the preoperative medication in patients for biliary surgery and only occasionally use Demerol. More advantageous is the use of a barbiturate 1½ hours before operation and atropine or scopolamine one hour preoperatively. *If pain is present, Demerol should be given at the same time as the atropine or scopolamine.* If the patient suffers from glaucoma, no belladonna derivatives should be used.

C. Factors in Selection of Anesthetic Agent

The choice of the anesthetic agent and *the method of administration* are determined by the physical status of the patient as well as the skill of the anesthesiologist. The most widely used anesthetic agents for biliary, hepatic and pancreatic surgery are (a) spinal, (b) epidural, (c) regional, (d) cyclopropane, (e) pentothal, nitrous oxide, oxygen and a muscle relaxant, (f) ether and oxygen. *These are not enumerated in order of frequency* of administration. Each agent and method has advantages and disadvantages which one has to evaluate carefully and individualize for each case.

1. *Spinal and Epidural Anesthesia*

Spinal *anesthesia can be administered in a single dose for shorter surgical* procedures such as cholecystectomy or choledochostomy. A longer acting anesthetic agent such as nupercaine or a mixture of pontocaine and dextrose with the addition of adrenalin is suitable *for the "single dose"* spinal anesthesia. *Good anesthesia can be obtained for two to three hours.* The chief advantage *of this method* is the extreme and consistent muscular relaxation, including a collapsed bowel, which is advantageous to surgical exposure.

If the contemplated operative time is longer than 2½ to 3 hours, or if it is uncertain, it is *preferable to use continuous* spinal anesthesia. This can be given through a catheter as described by Tuohy or through a malleable needle as described by Lemmon. The fractional administration of the spinal anesthetic agent permits regulation of dosage according *to the progress of*

surgery over long periods of time This technique is also used to great advantage in a "poor risk" patient to protect against too great or rapid fall in blood pressure The anesthetic agent is given in very small doses and the resulting anesthesia is segmental in character This prevents vasodilatation in the lower extremities and the resulting hypotension which may follow a single larger dose Spinal anesthesia is the best choice for patients who are jaundiced since it will not impose any additional metabolic burden upon an already damaged liver

Instead of the subarachnoid the epidural route may be chosen This technique is more complicated and has a greater percentage of failures In addition the volume of anesthetic agent injected is much greater with the epidural route The sympathetic fibers are affected to the same degree as with the subarachnoid injection and hypotension may develop possibly more profound due to the large volume of drug injected The subarachnoid space may be entered inadvertently and total spinal block with all its complications may develop

In cases where severe hemorrhage may be expected neither spinal nor epidural anesthesia should be used These patients would be deprived of their full power of compensation by the sympathetic paralysis accompanying spinal and epidural anesthesia General anesthesia administered by endotracheal route is the choice in such cases

2 Regional Anesthesia

Regional anesthesia is not very satisfactory for biliary tract or pancreatic surgery An abdominal field block is supplemented by a bilateral splanchnic block In almost all such cases nitrous oxide or cyclopropane inhalation anesthesia will also be required This adds all the dangers and possible complications accompanying general anesthesia to those of the local anesthetic agents It is sounder to start with a well planned and well conducted general anesthesia rather than to start with regional anesthesia and—after both the surgeon s and the patient s tolerance for inconvenience and discomfort have been taxed to the utmost—to supplement with general anesthesia The art of regional anesthesia is practiced extensively in France Their reported success depends largely upon a number of differences in the patients surgeons and the entire environment and atmosphere of the operating rooms It requires a great deal of time and patience on the part of the anesthesiologist as well as extreme skill and gentleness on the part of the surgeon

3 General Anesthesia

When general anesthesia is elected one should bear in mind that it is more difficult to obtain the same degree of abdominal relaxation as is produced by spinal anesthesia It is important to provide optimal conditions

for the surgeon so he may do his task in the shortest possible time Careful speed is of great importance, especially in poor risk patients and in radical operations The surgeon can operate with greater speed and gentleness if relaxation is good However the attainment of such *relaxation should not in any way endanger the patient*

With the use of general anesthesia relaxation may be obtained either by deep anesthesia or by the use of curare or curare like drugs Both methods have dangers inherent in the pharmacological action of the drugs used The danger of prolonged deep anesthesia especially in poor risk patients is obvious and needs no further discussion

With the use of curare and other relaxing agents the abdominal muscle the intercostal muscles and finally the diaphragm are paralyzed Unless assisted or controlled respirations are instituted immediately, grave consequences occur Carbon dioxide will accumulate if the respiratory exchange is inadequate due to curarization and the presence of two or three large gauze packs in the abdomen together with distended intestines and large retractors This carbon dioxide accumulation complicates the hypoxia due to insufficient exchange of gases In order to avoid this and produce good relaxation for upper abdominal surgery, the anesthetic agent is best administered through an endotracheal catheter with well assisted or controlled respirations The anesthesia should be in first plane of third stage The muscular relaxation is obtained by the use of curare like drugs Relaxation depends upon the interruption of the impulse on either the motor or sensory side of the reflex arc and also upon a free and unobstructed airway allowing for good gaseous exchange Even though the patient is heavily curarized abdominal relaxation may be poor because of excessive respiratory efforts due to respiratory depression or obstruction Balanced anesthesia is of great importance The patient has to be in the proper plane of anesthesia with the proper amount of curare and sufficient respiratory ventilation If all these requirements are met relaxation will be good and the surgeon can do his task with gentleness and speed

Some operations utilize an abdominal thoracic *incision* and the pleural cavity may be entered In such cases the presence of an endotracheal tube is essential Controlled respirations are better and safer when accomplished through a cuffed endotracheal tube Thus the dangers of inflating the stomach with air are eliminated

The selection of the agent used for the production of general anesthesia depends upon the status of the patient and the extent of the disease which is present Jaundice or other evidence of liver damage is often present in patients requiring biliary surgery

Ethyl ether is not the best choice since it decreases liver function and

bile secretion considerably It depresses reticuloendothelial cells, and glycogen decreases rapidly 50 per cent in the first hour and gradually after that (Adriani) Insulin production is decreased by ether anesthesia, and blood sugar may increase up to 200 per cent with a maximum rise during the first 15 minutes of anesthesia

Cyclopropane produces no change in liver function and no histological changes are reported (Adriani) The pancreas is apparently not affected and the blood sugar may be slightly elevated

Thio pentobarbital (sodium pentothal) has little effect on liver and pancreas It raises the blood sugar slightly

Nitrous oxide given with adequate amounts of oxygen has no effect upon liver or pancreas A very excellent method of anesthesia for operative cholangiography under pentothal, nitrous oxide, oxygen and a short acting curare like preparation has been described by Bernstine and Golden

D Management of Anesthesia for Biliary Surgery

When spinal epidural or regional anesthesia is used it may be supplemented with light general anesthesia This will add to the comfort of the patient and overcome some of the traction reflexes Oxygen by mask or catheter should always be administered in upper abdominal work regardless of the type of anesthesia

Five per cent dextrose in water should be given intravenously and blood should be replaced as it is lost Blood pressure pulse and respirations are carefully watched because from these one can foretell impending shock and take the necessary steps to prevent it

If biliary obstruction has been present and it is relieved during operation one may see a serious drop in blood pressure This is due to additional blood rushing into the liver to fill the previously compressed capillaries with pooling in this organ thus reducing the circulating blood volume It is best combated by increasing the speed of administration of blood If the drop in blood pressure is alarming and prolonged and cannot be corrected by administration of blood a vasopressor drug may have to be used However this should be done only after adequate amounts of blood have been given

If general anesthesia is used the same precautions are taken There is one added factor which is the maintenance of a clear and unobstructed airway If cholangiography is done during the operation under general anesthesia respirations must be stopped during exposure of the films The apnea present with the use of controlled respirations can be used to great advantage If the patient is under spinal or regional anesthesia in combination with very light general anesthesia manual hyperventilation for a few seconds prior to compression of the breathing bag will suffice in most cases

If the patient is obese and difficult to keep in the first plane of surgical anesthesia, the use of a short acting muscle relaxant may be used to produce the apnea

F Prevention of Postanesthetic Complications

Prevention of postanesthetic complications begins with proper preparation of the patient for the contemplated surgery This is the surgeon's responsibility to institute and the anesthesiologist's to evaluate A high carbohydrate diet including parenteral administration of glucose may protect hepatic cellular function Fluid and electrolyte balance should be normal Preoperative administration of compatible blood is necessary to correct anemia and nutritional deficiencies with or without hypoproteinemia The use of blood during operation with a patient under general anesthesia should be limited to *compensation for blood loss It is not given for "prophylaxis"* Transfusion reactions such as chills or urticaria are not apparent under general anesthesia and more severe reactions may follow When the patient is not anesthetized, transfusion reactions are not masked and can be treated properly by rapidly discontinuing the administration of blood and by the intravenous use of antihistaminics, 1/6 molar lactate and other therapy as indicated

The causes of postoperative morbidity and mortality have changed during the past 18 years (Glenn et al) The mortality rate has been sharply decreased Today the largest single cause for death following biliary tract surgery in non malignant diseases is hepatic insufficiency Pulmonary complications have become uncommon (chapters 6 and 9) Postoperative peritonitis, intra abdominal abscess and fistula formation are prevented by the use of antibiotics An increased tendency of degenerative diseases such as diabetes, arteriosclerotic cardiovascular disease and hypertension has been shown Proper selection of anesthesia and skillful conduction *of it will do* much to provide a smooth postoperative course Many *so-called postoperative* complications are in direct relation to the anesthesia and are caused either by poor selection of the agent or by poor management

The recovery room plays a very important part in the prevention of *postoperative complications* In it the patient is under the care and observation of a specially trained graduate nurse with direct *supervision* by the anesthesiologist

If general anesthesia has been used the trachea is repeatedly aspirated by suction in order to avoid aspiration of mucus or gastric content *into* the bronchi Nasogastric and other drainage tubes are observed for proper function Blood *pressure* pulse and respirations are checked repeatedly Intravenous fluids various supportive drugs and oxygen are administered

as indicated Patients remain in the recovery room until all reflexes have returned and until they respond to questioning

Patients who have had spinal or regional anesthesia remain in the recovery room until blood pressure and pulse rate are stabilized In order to avoid post spinal headache the patients are instructed to remain flat in bed for six to eight hours In addition fluid intake is increased 1000 cc per 24 hours above the normal intake if there is no medical or surgical contraindication

The recovery room has facilities for bronchoscopy Tracheotomy and cardiac resuscitation trays are sterile and available for use at all times Any emergency arising in the immediate postoperative period can be dealt with efficiently in a well equipped well staffed recovery room and many post operative complications and fatalities can be avoided

I Treatment of Pancreatic Pain by Analgesic Blocks

Severe pain is an outstanding symptom of pancreatitis and necessitates immediate relief Morphine sulphate and other opiates are unsatisfactory because they induce smooth muscle spasm of the biliary and pancreatic ducts and of the duodenum and thereby produce or increase pain in biliary tract disease Demerol is the drug of choice because it is a mild spasmolytic

Analgesic blocks can be used to great advantage In order to discuss the treatment of pancreatic pain by the interruption of sensory and sympathetic fibers it is important to understand the origin and conduction of pain from this organ to the central nervous system According to Richins the receptors are probably associated with blood vessels in and around the pancreas These are considered to be terminals of visceral afferent fibers They go through the splenic pancreaticoduodenal and celiac plexus the greater lesser and least splanchnic nerves and then through the lower thoracic and upper lumbar paravertebral ganglia Via their white rami communicates they proceed to the posterior spinal nerve roots Here the visceral and somatic afferent fibers join and enter the spinal cord (Hollinshead)

The segments involved are from the sixth thoracic to the first lumbar segment bilaterally although the left side is predominant

Analgesic blocks for pancreatitis not only relieve pain almost instantaneously but also minimize shock by combating the systemic effects of pain (Bonica) Concommitantly the efferent sympathetic fibers are blocked overcoming reflex spasm of the duodenum sphincter of Oddi and the entire ductal system leading to emptying of dilated ducts of the biliary tract and pancreas (Gage) Vasodilatation occurs with improvement of blood supply and lymphatic drainage thus decreasing interstitial edema which is present

in almost all cases If paralytic ileus is part of the picture of acute pancreatitis, this too is mitigated by sympathetic paralysis

Thus we see that analgesic blocks have a much wider scope of action than just relief of pain

Four different types of analgesic blocks may be used

1 Continuous peridural block is a very excellent technique in establishing a bilateral somatic sensory as well as sympathetic efferent and visceral afferent block with only one puncture The results have been very gratifying even though the technique is not as simple as some of the other blocks

2 Splanchnic block is widely used It has the advantage that it also blocks the vagus fibers going through the celiac plexus (Bonica) The vagus is responsible for transmission of impulses to the secretory portion of the pancreas It is a relatively simple procedure and can be done in acutely ill patients The disadvantages are that in most cases it has to be done bilaterally and that it may lead to considerable drop in blood pressure due to the sympathetic paralysis causing widespread vasodilatation of the lower extremities and the abdominal viscera A polyethylene catheter may be introduced and the local anesthetic agent may be given by repeated single injection or by continuous drip of a more dilute solution

3 Paravertebral block may be used for the treatment of pancreatitis The sixth to the twelfth thoracic segment has to be blocked bilaterally, which causes the patient considerable discomfort Since most of the sympathetic fibers are paralyzed in this procedure a considerable drop in blood pressure may occur However, this occurs rather rarely This type of block is used to great advantage when pain is well localized and does not involve a large area

4 Subarachnoid block may be performed with a dilute solution of local anesthetic agent This will result in interruption of sympathetic fibers as well as visceral afferent and possibly somatic sensory fibers depending upon the strength of solution Motor fibers are spared by dilute solutions

Analgesic blocks for the treatment of pancreatitis are only part of the therapeutic regime which is available to us (chapter 5) It is important to establish fluid and electrolyte balance and treat shock if such should be present

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11

CHOLEDOCHOTOMY

A Technical Considerations

Many incisions are used to expose the bile ducts Vertical and transverse incisions are each advantageous depending upon the contour of the costal margin (fig 64) Transverse subcostal (Kocher) incisions usually furnish both adequate approach and wide exposure together with fewer postoperative eventrations However they do not always permit sufficient exposure of the pancreas Longitudinal incisions give adequate exposure to the gastric and pancreatoduodenal regions but there may not be sufficient exposure of the liver bed Modifications and extensions of basic incisions are optional with the surgeon in deference to the patient s individual characteristics

Baronofsky and Carlock advocate that the surgeon operate from the left side of the patient This permits the assistant to retract the right costal margin and liver edge Another assistant can retract the colon and maintain suction or whatever sponging is necessary From the left side of the patient the surgeon may readily use his left hand for control and exposure of the foramen of Winslow and the blood supply Our experience has been that dissection of the cystic duct and common duct landmarks is greatly facilitated in this manner Cholecystectomy however is awkward

1 Incisions (Fig 64)

a Paramedian

In upper 60 per cent of abdomen on the right for biliary tract and pyloroduodenal disease and on the left for gastric and pancreatic disease May extend from the xiphoid—about 2 cm from and parallel to the midline—to below the umbilicus Excision of the xiphoid will remarkably increase exposure Fibers of the rectus muscle are retracted laterally Avoid superimposition of incisions in anterior (more lateral) and posterior (more medial) rectus sheaths

b Trans rectus

As in paramedian (*a*) At apex of incision insertion of rectus muscle or costal margin may be cut and xiphoid also resected to increase extent of exposure Suture ligation of the external epigastric artery (or branches) is frequently required This incision preferred to the paramedian

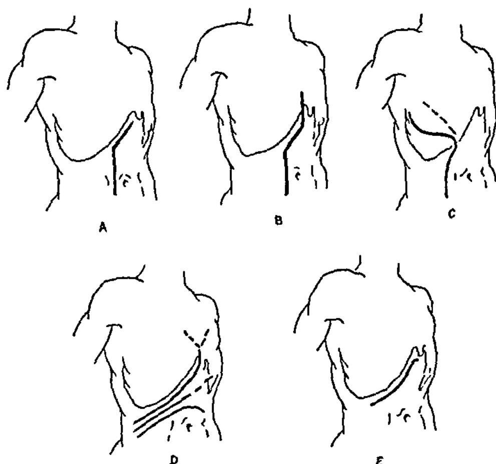


FIG 64 ABDOMINAL INCISIONS USED FOR BILIARY TRACT SURGERY

Vertical incision (a) paramedian (b) transrectus and (c) pararectus with extensions through intercostal space or across the costal cartilages

Transverse incisions (d) right upper with extensions 1) through midline and para xiphoid 2) across into the left chest or 3) across the entire abdomen and (e) para costal or subcostal

c Lateral Rectus

Primarily intended for exploration and to be extended medially and obliquely or inferiorly (abdomen) or laterally across costal cartilage (thoraco abdominal) On the right for biliary, hepatic or renal disease and on the left for gastro-esophageal pancreatic or splenic disease

d Transverse (Upper Right)

Utilized for biliary tract, pyloroduodenal and right colon lesions in patient with short abdomen and wide costal plane From midway between xiphoid and umbilicus laterally to midway between costal margin and crest

of ilium Muscle splitting (or cutting) laterally Cut right rectus muscle, retract left rectus laterally May be extended into left flank (for pancreas or colon) into left thorax for high gastric lesions, pancreas or spleen resection It may also be extended to the right thorax for greater exposure of the liver

c Subcostal

Upper abdominal in patients with very wide costal plane incision in line of either oblique muscle extending into rectus sheath but without cutting rectus muscle which is retracted laterally (a modification of the transverse incision) a painful incision

2 Instruments and Materials for Biliary Tract Operations

a Retractors

1 Balfour self retaining retractor
6 Deaver retractors medium or wide blades (short medium and long) Kelly or Richardson retractors are optional in lieu of Deaver retractors
2 Harrington retractors small and medium
1 Reverdin abdominal spatula
2 Volkmann three pronged retractors sharp or dull
4 lengths of stockinette as retractor covers

b Forceps

12 Backhaus towel forceps
2 Bozeman dressing forceps
2 Foerster sponge holding forceps 9¾ inches
1 Splinter forceps 3½ inches
4 Thumb forceps 5 7 and 9 inches
2 Tissue forceps 1 and 2 teeth 5 and 7 inches

c Hemostats

6 Allis 4 and 5 teeth
8 Allis intestinal
4 Babcock
6 Expanded tip dissection hemostats (Gray Behrend)
12 Mayo Pean Rochester Pean or Kelly curved hemostats 6 inches
4 Mayo Carmalt hemostats 8 inches
6 Moynihan (or Mixter) hemostats
6 Ochsner hemostats 7¼ inches
14 Straight hemostats

d Needle Holders

2 Hegar needle holders 7 inches
2 Mayo or Crile needle holders 5½ inches

e Cutting Instruments

2 Bard Parker knife handles # 4
1 Bard Parker knife handle # 7
1 Bard Parker knife handle # 3
Scalpel blades to fit above
2 Mayo curved on flat scissors 5 and 7 inches
2 Mayo straight scissors, 5½ inches
1 Metzenbaum scissors 5½ inches
1 Metzenbaum scissors 7 inches
1 Suture scissors
1 Wire cutter

f Common Duct Instruments

1 Set Bakes dilators (1 mm to 8 mm sizes)
2 Blake gallstone straight and curved forceps
4 Cystic duct plain jaw forceps
1 Desjardins gallstone forceps
1 Luer Koerte gallstone size 4 scoop
1 Moynihan gallstone probe and scoop
1 Moore gallstone scoop
1 Ochsner gallstone spiral probe 14 inches long
4 Stone forceps (straight and curved)
1 Set lachrymal duct probes
1 Flexible plain long probe
1 Uterine sound small
1 Ureteral sound
1 Ureteral dilator
1 Ureteral stone grasper

g Injection Material

Trocar and Cannula medium or small size
20 cc syringes (3)
Aspirating needles gauge 18 (3 inches) and gauge 15 (4 inches)
Hypodermic needle and angled needle gauge 23 B D # [illegible]
Ureteral catheters size 6–10
Urethral catheters size 8–12 20–22

Polyethylene catheter
T tubes, short and long sizes 16–22 ("Best" type)
T tubes (Cattell) with "Y" for use in duct anastomoses
Special "T" drains
Asepto syringe
Irrigating can with antrum tip and suction tubing
Triumph syringe
40 cc Diodrast (35 or 70%) and file for cutting ampoule
50 cc 1% novocain
500 cc saline (warm)
Manometer (water) with three way stopcock

h Other

2 Deschamp aneurysm needles
Long knife handles right and left angles
Periosteal elevator
Costatome
Rib spreaders
Rib approximators
Cameron light
Nerve clips
Probe with eyelet
Grooved director
Poole abdominal suction tip and tubing
Suction tip (neurological)
Curette double end
Oxycel Hemopac or Gelfoam
Bovie unit with coagulation tips
Cardiac defibrillator
Pott's clamp 2 sizes

i Drains (in Addition to "T" Drains)

Soft black rubber tubing 10 inches long various diameters
Carrel (or Dakin) tubing 12 inches long
Penrose drains wide and narrow with removable wick
Pezzer catheters closed (or open) top sizes 12 20 24
Robinson catheter sizes 10 14 and 18
Malecot catheter with wings size 14
Mercier curved stylet
Metal sump (Babcock) 6 and 8 inch
Plain folded packing 2 inches wide

j *Suture Materials*

(1) Ligation and suturing
Catgut chromic #0 or #00
Silk #000 or #00
Atraumatic #000 (chromic and silk)

(2) Cystic pedicle ligatures
Catgut chromic #00
Silk #00

(3) Anastomosis
Catgut chromic #00
Silk #000 and #00

(4) For hemostasis
Catgut plain #000, #00
chromic #00 #0 and 1
atraumatic catgut sutures, chromic 00 threaded on curved and straight intestinal needles

(5) For closing
Stainless steel or tantalum wire #000 or #0 or #1 for tension sutures
Dermalon #3 for tension sutures
Dermalon #000 for skin closure
Skin clips (Michele)

k *Needles*

4 Ferguson ½ circle curved taper point #10 and #16
2 Keith abdominal straight 2½ inches
1 Wylie pedicle
2 Mayo ½ circle curved trocar point #2
6 Mayo ½ circle curved taper point, two of each size #3 and #5
4 Murphy ½ circle curved taper point #3 and #4
2 Regular surgeon's cutting edge ⅜ circle curved #3 and #8
2 Regular intestinal ½ circle curved taper point 1 inch
2 Straight intestinal 2 inches (taper point)

3 Operative Procedure

a *Laparotomy (Oblique or Transverse Incision)*

(1) Skin incision is made to the fascia Hemostasis is obtained
(2) Superficial fascia sutured to folded towel by running lock stitch
(3) External oblique fascia is incised aponeurosis is split and retracted Internal oblique and transversalis muscles are split and retracted protecting nerves
(4) Rectus sheath and muscle are incised Suture ligatures to bleeding

points include rectus sheath Superficial epigastric artery ligated separately

(5) Retractors elevate the abdominal wall Peritoneum is exposed and mobilized, grasped and incised Opening in the peritoneum is extended Viscera protected by continued elevation of abdominal wall and by guard to cutting instrument Adjacent and underlying adhesions are liberated by sharp and blunt dissection Peritoneal cavity is explored

(6) Definitive operation is done

(7) Hemostasis is secured sponges are counted

(8) Peritoneum and fascial layers closed in tiers Skin sutured Dressing applied

b *Cholecystectomy*

(1) Hepatic flexure mobilized covered with moist pad and retained caudal by retractor

(2) Pyloroduodenum mobilized to the left Moist pad applied beneath retractor to produce tension on the duodenum

(3) Adhesions between the gall bladder, liver and adherent viscera separated clamped incised and ligated

(4) Abdominal pads are replaced and retractors reinserted to maintain exposure (fig 65) Small marked sponge inserted in the foramen of Winslow

(5) Common bile duct and hepatic pedicle (hepatoduodenal ligament) palpated for evidence of abnormality Hepatic artery and portal vein identified (fig 66)

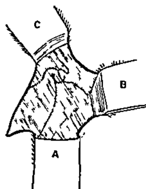


Fig 65 Exposure of Bile Ducts—Retraction

Three principal directions for retraction are established (a) downward mobilization of hepatic flexure (b) left lateral retraction of pyloroduodenum (c) upward displacement of right lobe of the liver

(6) Dome of liver is palpated and air permitted into right diaphragmatic space

(7) Fundus of the gallbladder is clamped Gentle traction is applied upward and forward Small sponges placed to left of gallbladder overlying right lobe of liver Retractor placed on this sponge to expose left side of the gall bladder and the common hepatic duct

(8) When the gallbladder is distended the fundus is grasped incised and a trocar aspirates it At this time large calculi may be removed in order to improve visibility at the cystic pedicle The gallbladder opening is secured by application of Kocher forceps or figure of 8 suture

(9) The wall of the gallbladder close to the cystic duct is clamped Slight traction is exerted by this clamp to aid the dissection

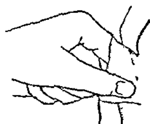


FIG 66 EXPOSURE OF BILE DUCTS—PALPATION

Hepatic pedicle is palpated to identify abnormalities of or within the bile duct the location of the hepatic artery and the contours to the superior aspect of the pancreatic head With the left forefinger in the foramen of Winslow the thumb can palpate and if necessary compress the hepatic artery

(10) Ventral and dorsal peritoneal reflections from gallbladder to common duct and duodenal wall are incised to expose the pedicle of the gallbladder (fig 67)

(11) The neck of the gallbladder the cystic duct and the common duct above and below the junction with the cystic duct are visualized (Cholangiogram or choledochostomy may be done)

(12) Dissection of hepatic duct continued toward hilum of liver Cystic artery is identified It is doubly ligated close to the gallbladder wall The artery is divided (fig 68a)

(13) Cystic duct is freed and ligatures placed Ligature closer to the gallbladder is tied The other is not tied if cholangiogram is to be done

(a) Cystic duct incised by optional incision Ureteral catheter is passed into the common duct as far as it will go and then is withdrawn about one inch Polyethylene catheter is useful (Block)

(b) Fluid is injected through the catheter to determine patency of papilla and flow into the duodenum distensibility of the common duct and status of the proximal duct system
(14) Cystic duct ligated (fig 68b)

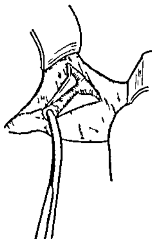


FIG 67 EXPOSURE OF BILE DUCTS—DISSECTION

Cholecystic choledochal membrane is incised while traction is maintained laterally on the gallbladder and medially on the pyloroduodenum Di section of the antero lateral hepato duodenal ligament exposes the cystic duct union with the choledochus

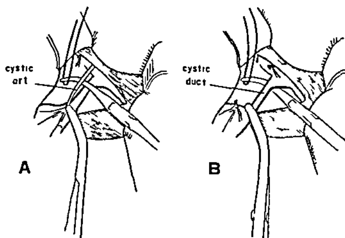


FIG 68 LIGATION OF THE CYSTIC PEDICLE

A A ligature is pa ed around the cystic artery This is tied close to the gallbladder to avoid damage to the hepatic artery

B A ligature is passed around the cystic duct A second ligature may be tied loosely to permit intubation of cystic duct for cholangiography

(15) Cystic duct stump and "ampulla" of the gallbladder elevated Incise peritoneum along sides of the gallbladder wall attachments to liver (fig 69a) Sharp scissors and blunt finger dissection is accomplished behind and close to the gallbladder Bleeding vessels and *open bile ducts* in liver bed are clamped and ligated

(16) Gallbladder dissection from liver bed completed leaving *hemostat* on gallbladder attachment at apex of liver as traction guide Raw

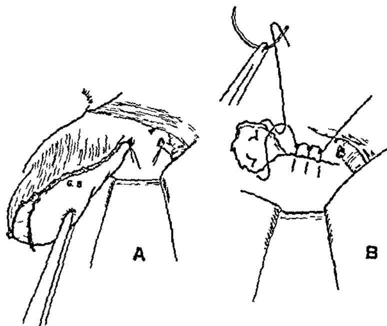


FIG 69 REMOVAL OF THE GALLBLADDER

A Gallbladder peritoneum is incised sharp and blunt dissection against the gall bladder wall mobilizes the gallbladder from its liver bed

B Liver bed is lock sutured Separate bites of needle taken into peritoneal edges and into liver bed to eliminate potential "deadspace"

surfaces in the liver fossa left by removal of the gallbladder closed by suturing peritoneal reflections to the defect (No 'dead' space is left) (Fig 69b)

(17) Penrose drain is placed to region of foramen of Winslow

c *Choledochostomy (Vide infra chapter 11 Section B)*

(1) Hepato-duodenal ligament cleared for at least 2 cm from common hepatic and bile ducts (To improve exposure of common bile duct tension on duodenum or incision of duodenal peritoneal reflection may be needed) (Fig 71) Duct is aspirated to obtain bile (to differentiate from portal vein and hepatic artery) Anastomotic

vessels crossing antero lateral aspect of the common bile duct are ligated

(2) Stay sutures placed in medial and lateral superior aspects of the bile duct (fig 72) Longitudinal incision (1 cm long) is made in the bile duct wall between the sutures Stay sutures may be replaced (instruments are not applied to duct wall) Duct incision may be lengthened if desired

(3) Small urethral (or ureteral) catheter is inserted into distal common duct as far as possible and then withdrawn 1 to 2 cm (fig 73) The duct is palpated along catheter Warm saline is irrigated into catheter and return flow aspirated by suction tip on sponge in the foramen of Winslow

(4) Another catheter is inserted into the proximal common duct for a short distance Palpation may be attempted along the indwelling catheter Warm saline is irrigated

(5) Catheters are removed and flow from duct is inspected If calculus is suspected a (stone) forceps is inserted (The Bakes dilator, 1

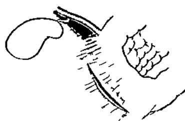


Fig 70 Mobilization of Duodenum—I

Incision into lateral duodenal peritoneum permits mobilization of duodenum medially to expose retroperitoneal duodenum pancreas and common bile duct



Fig 71 Mobilization of Duodenum—II

On some occasions the entire lateral leaflet of peritoneum to the duodenum is incised Low implantation of the papilla may require this procedure

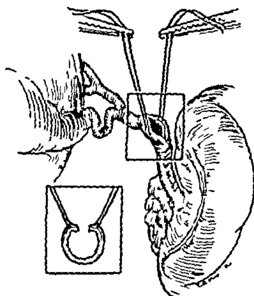


Fig 72 Choledochotomy—I

Traction sutures are inserted along the anterosuperior aspect of the duct Longitudinal incision for 1 5 cm is made into the duct while the anterior duct wall is elevated to avoid incision through both walls of the bile duct

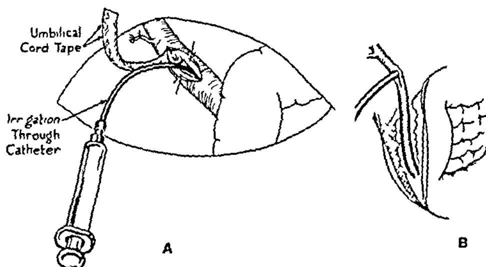


Fig 73A Choledochotomy—II

A catheter is inserted for irrigation of the distal bile duct Umbilical cord tape is packed into the proximal bile duct to prohibit regurgitation into the hepatic ducts from the lavage A similar procedure is accomplished for irrigation and exploration of the hepatic ducts

Fig 73B Choledochotomy—III

The duct is closed around a "T" drain Sutures are placed distally No sutures are placed proximally (This may be reversed)

or 2 mm size may be passed under finger tip guidance)

(6) T tube placed into duct, duct edges closed Stay sutures not utilized for closure but remain in place until after a cholangiogram (fig 73b)

(7) Cholangiogram is taken Duct is reopened if indicated for removal of calculi instrumentation or further irrigation T tube replaced Repeat cholangiogram as often as indicated Duodenum may be opened for examination of the papilla of Vater

d Choledochostomy as a Reoperation (*Vide infra Chapter 11 Section C*)

(Abdominal incision and exploration are done as above (a) except that in the presence of a biliary fistula (or tube) the tract is "cored" to the common bile duct Vertical (longitudinal) incision is lateral to previous operative sites in order to avoid adhesions)

(1) Identify inferior and lateral aspect of right lobe of liver

(2) Mobilize antero-lateral structures including the hepatic flexure medially and caudally It may be necessary to identify a free area to the right of the ascending colon

(3) Dissection proceeds antero-superiorly to identify the triangular ligament of liver as superior limit to dissection

(4) Mobilize pyloric antrum and adjacent duodenum to the left and slightly caudal

(5) Dissect along liver margins to hilum staying over or to the right of the gallbladder fossa if possible Approach liver hilum from these three aspects

(a) from lateral to medial
(b) from hilum downward
(c) from pyloroduodenum forward

Use tension on mobilized structures and sharp (scissors) dissection spread (ligate or clamp and ligate) then cut

(6) When second and third portions of duodenum are identified the lateral peritoneal reflection may be incised The duodenum is mobilized antero medially This adds a fourth plane to dissection this aimed from posterior plane anteriorly

(7) The abnormal common duct is palpated The duct wall may be thick may contain definite calculi or may be dilated (The value of an indwelling tube or persistent fistula is inestimable) Aspirate any suspicious area with needle for more positive identification

(8) Stay sutures (atraumatic silk 000) placed along chosen lateral and antero medial aspects of the common duct Sutures are placed firmly and are aimed from the left foot to the right shoulder of the patient

(9) Sponge has been placed in foramen of Winslow Dissect and clear 2 cm segment of the bile duct As soon as possible, foramen of Winslow is cleared to permit digital control of the blood supply
(10) Longitudinal incision is made into common bile duct and contents permitted to escape Any indwelling tube is removed and fibrotic edges of the duct defect are freshened
(11) Double intubation (proximal and distal ends) of bile duct is done with (urethral or ureteral) catheters Saline solution is irrigated Instrumentation may be done with blunt stone forceps to remove calculi A small (1 or 2 mm) Bakes dilator may be passed under guidance of very light finger tip pressure
(12) ' T ' tube placed, duct closed and cholangiogram done Remove ' T tube if necessary to correct residual abnormalities
(13) Optional procedures (Chapter 12)
   (a) Long area of stricture is excised (more than 1 cm) and repaired by end to end suture
   (b) Short area of stricture (less than 1 cm) is incised longitudinally and sutured transversely
   (c) Distal common bile duct is ligated Proximal common bile duct is anastomosed to jejunum (loop or limb)
   (d) Lateral anastomosis between incision into common bile duct and adjacent duodenum
   (e) Concurrent duodenotomy
(14) Additional incision is made through normal duct, above or below anastomoses to permit external limb of T tube to emerge May use ureteral catheters
(15) Hemostasis is secured Operative area is inspected to obviate future sequellae from duct or bowel obstruction Gastroenterostomy may be indicated if there be danger of duodenal obstruction
(16) Stab wound incision made for emergence of "T" tube Penrose tube drain is not usually used If absolutely indicated, any additional drain is kept away from the "T" tube area

B Choledochotomy at the Time of Cholecystectomy

1 Indications for Choledochotomy

Decision to open the common bile duct is usually made because of the history and laboratory evidence indicating obstructive jaundice However most patients with calculous cholecystitis should be prepared as if for common duct surgery Because of this cholecystectomy should be elected to avoid inflammation or edema which can obscure anatomical landmarks

Careful, thorough surgery on the common duct will not add to the post

operative morbidity In fact, the necessity for secondary operations on the biliary duct may often be avoided

There are six major groups of patients in whom the common duct should be explored at the time of original cholecystectomy 1) Cholelithiasis with a stone palpable in the common bile duct 2) Cholelithiasis when the common bile duct is thickened and dilated to more than 1 cm in diameter 3) Cholelithiasis where the stones are small or friable and where the cystic duct is dilated 4) In the absence of cholelithiasis evidence that the pancreas is enlarged edematous or indurated, or where there is evidence of fat necrosis and/or pancreatic abscess 5) Unexplained biliary tract symptoms particularly in the absence of cholelithiasis 6) In other cases with abnormal anatomical findings a palpable mass in or near the common bile duct obstructive jaundice without obvious cause and in some older age group patients who have had biliary tract disease of long duration

2 Duct Exploration without Incision

One method for exploration of the common duct is done without surgical incision Examination of the duct may be done through the cystic duct as follows (a) Palpation (b) Passage of catheter through the cystic duct and palpation over the catheter (c) Irrigation of the duct system (d) Injection of radio-opaque media through the cystic duct followed by cholangiogram

In one third of cases the common bile duct is transpancreatic for 2 to 4 cm and will not be palpable or visible (fig 7c) In another third of cases the common bile duct will be adjacent to both the pancreas and the duodenum (fig 7b) It can be palpated and may be visible just beneath the fascial reflections of the pancreatic capsule This covering may be incised with due regard to the venous tributaries overlying it In the other third of ca es practically the entire retroduodenal common duct is visible or easily exposed (fig 7a)

The common duct may be explored simultaneously by means of a catheter in the cystic duct and by duodenotomy

3 Choledochotomy

A second method for exploring the common bile duct is through longitudinal or oblique incision into the common hepatic or common bile duct (fig 72) The incision in the duct may be 1 or 2 cm long

Observations can be made by passage of (a) an ureteral catheter (b) an urethral catheter (c) metallic probes (d) or choledochoscope Fluids are injected to test patency of the papilla and pressure within the duct Additional information may be gained by the presence of a nasal tube in the duodenum Injection of weak hydrochloric acid or magnesium sulfate solution therein may provide additional facts particularly during cholangiography

Observations by one method alone may not be entirely valid for evaluating the common duct It is best to rely upon multiple methods These include (a) palpation over indwelling (ureteral) catheter, (b) cholangiography either through the cystic duct stump or the common duct, and (c) the pressure of injected fluids and the rate of flow into the duodenum It is essential that the observations be evaluated overall since each individual method has some inherent error

For example radiography may not demonstrate a stone to be present because the stone may be hidden by the opacity of the dye On the other hand, entrance of fluid into the duodenum may be obstructed temporarily by spasm at the papilla

Doubt may exist about the normalcy of a common bile duct during operation even after irrigation manipulation, palpation, radiography and reexamination Such cases include those in which (a) small calculi have been removed from the duct, (b) flow *from the common duct into the duodenum* is delayed, (c) suspicious defect is seen on operative cholangiogram at the termination of the common bile duct—excess length or deformity of the transduodenal common bile duct, (d) duodenal diverticulum is adjacent to papilla of Vater (e) there is a penetrating duodenal ulcer, (f) there is enlargement nodularity or deformity in the papillary region which may involve duodenum pancreas or papilla

Under such circumstances a third method is utilized This is the transduodenal exposure of the papilla together with exploration through the open common bile duct Duodenotomy does not per se contribute to morbidity (chapter 12)

4 Evaluation of Choledochotomy

Incision into the common duct is required

a by the appearance of the abnormal duct which is
   (1) greater than 1 cm in diameter
   (2) thicker
   (3) indurated,
   (4) contains calculi
b in the presence of clinical states which have indicated probable chronic disease in the duct (obstructive jaundice pancreatitis tumor),
c and in certain carefully considered situations of justifiable clinical suspicion

Morbidity and mortality rates due to opening a normal common bile duct are insignificant when unsuspected choledocholithiasis is relieved (2 per cent unsuspected) or a tumor of the biliary tract is eradicated

In a recent survey of 677 patients from three institutions over a 7 year period it was found that the common bile duct was explored in 35 per cent

of patients at primary cholecystectomy In these the overall mortality rate was 1 9 per cent Patients having only cholecystectomy had a mortality rate of 0 5 per cent Patients having *common duct exploration (including* secondary operations) had a mortality rate of 3 6 per cent Patients who had reoperation or later fatalities since cholecystectomy cannot be satisfactorily enumerated statistically However, these are more frequent than suspected By doing the common duct exploration at the time of original cholecystectomy there is no doubt but that more favorable conditions will produce fewer post cholecystectomy complications

5 Precautions

It is essential to avoid damage to the hepatic pedicle Carelessness, over confidence and haste are as culpable in accidental trauma as anatomical ignorance anomalies or disease In order to keep complications to a minimum the following mnemonic "vowel check list" is constantly repeated for the operator s residents anesthetist s and patient s benefit

A Adequate proper anesthesia with sufficient relaxation

E Exposure sufficient for each problem additional length of incision additional assistants sufficient lighting including lighted retractors efficient suction and plenty of time

I Identify all structures cystic and hepatic arteries cystic hepatic and common bile ducts tension may be required on pyloroduodenal region for dissection of duodenal hepatic ligament (hepatic cystic pedicle)

O Overlook nothing anomalies may be anywhere accessory ducts and vessels are in a 12 per cent group of patients but the patient does not know which group he joined

U Undue tension is avoided during ligation of the cystic duct and artery

Hemorrhage from cystic vessels may occur with devastating suddenness during cholecystectomy There is one safe way to deal with the crisis The left index finger is placed through the foramen of Winslow to compress the hepatic artery (in the edge of the gastrohepatic ligament) against the left thumb (fig 66) This should stop all arterial bleeding in the gallbladder area The field is cleared by aspiration and sponging The vessel is then clamped with a hemostat and ligated accurately The bleeding point is accurately observed to avoid injury to the ducts and hepatic artery

Common duct injury may also result because of inadequate visualization of the junction for cystic and hepatic ducts When the dissection has been scanty and the field is deep—moderate excess or sudden traction on the cystic duct may bring the anterior common duct *wall to within the loop* of ligature The common duct is thereby distorted narrowed and may be obstructed

If this damage is recognized it can be immediately remedied The defect in the common duct wall can be repaired after removal of the ligature by one of several methods A "T" tube can be placed through the opening at the base of the cystic stump The opening may be closed transversely with "T" tube drainage through another site in the common duct The damaged segment of common duct may be excised and end to end anastomosis of the adjacent portion of duct done

The surgeon may clamp the hepatic artery proper or one of its branches (other than the cystic) without being aware at the time that he has damaged an important blood vessel Subsequent liver necrosis may be fatal

For more facility in exposure of structures in the triangle of Calot and in providing efficiency in careful dissection, the surgeon may stand to the patient's left This position however, is disadvantageous for cholecystectomy

C Secondary *Operations on the Common Bile Duct*

1 General

One or more years following cholecystectomy, in approximately 5 per cent of patients, reoperation may be required on the biliary tract because of pain, jaundice chills, fever or other symptoms Statistics are difficult to evaluate because the patient who has postcholecystectomy discomfort does not often return to the same surgeon (chapter 15)

Ninety per cent of patients who require reoperation have residual choledocholithiasis *Ten per cent of these have intrahepatic calculi* Other causes for reoperation on the common bile duct are related to defects at the cystic duct stump (such as a large remnant which may contain calculi neuroma or pseudocholedochus cyst) and to disease in the common duct (including stricture stricture with fistula, cholangitis, pancreatitis or tumor)

Biliary cirrhosis and portal cirrhosis may frequently be present because of the many attempts to palliate a "postcholecystectomy syndrome under medical regimen Medical therapy should not be prolonged when obstruction to *bile flow is evident*

Reoperation on the biliary tract should not be done unless the patient has full thorough evaluation of and management *for extrabiliary tract* disease Such may produce symptoms representing the postcholecystectomy syndrome or may represent the original symptoms *for which the cholecystectomy* had been done These include peptic ulcer hiatus hernia colonic diverticula or malignancy arthritis neuralgia, chemical idiosyncrasies or dyspepsias and psychoneuroses (Chapter 15)

Adequate clinical laboratory and radiologic examinations are essential

Hepatic renal and cardiovascular systems are fortified Intestinal alimentation and antiseptics are administered Nasogastric or a long intestinal tube may be passed, particularly if gastric dilatation or ileus be present

It should further be stressed that the majority of these operations cannot be completed in less than three hours Therefore the best care is required in selection of anesthesia In addition adequate supply of blood for transfusion is required

2 Method for Exposure of the Duct

Because of frequent dense adhesions in the gallbladder bed, a second approach to the area should be through (comparatively) uninvolved structures If previous surgery has been through a right rectus incision the second procedure may well be a pararectus or transverse incision It is preferred to enter the peritoneal cavity in an area which is relatively free from adhesions

Sharp dissection is utilized to clear superficial adhesions Approach to the hepatic pedicle is made from several directions

The hepatic flexure of the colon is identified and freed Dissection is continued to mobilize the descending limb of the duodenum by incision of the lateral peritoneal reflection The mobilization should carry the duodenum and the attached head of the pancreas anteromedially away from the posterior abdominal wall and retroperitoneal fascia

At this point dissection is directed to mobilize the stomach and pyloric region away from the liver hilum and hepatic pedicle The triangular ligament and the gastrohepatic omentum may form a dense impenetrable vascular mass Dissection is directed against the liver edge working downward and from medial to lateral direction into the interlobar hepatic cleft towards the gallbladder bed Just as soon as dissection becomes sanguine and difficult it is discontinued to begin the third route to the hilum (The *importance of establishing the previous dissections* is now evident since retraction gently applied on the stomach and on the duodenum to the left and upward and on the colon downward and to the left permits optimal tension on the hepatic pedicle Dissection is simplified when tissues are under tension)

The common bile duct is anterolateral to most other structures in the hepatic pedicle It is the superior and lateral border to the foramen of Winslow This foramen is often obliterated but as the duodenum is mobilized medially the foramen may be visualized Between the foramen and the liver hilum lies the promised land

Patients with obstruction to the bile ducts may manifest a dilated bulbous duct A mobile stony tissue can be a calculus a neoplasm or a lymph node The portal vein or hepatic artery may be camouflaged An aberrant

pancreatoduodenal branch of the gastroduodenal artery may cross in an unexpected plane This dissection is a most difficult procedure for the surgeon

Once bile is identified and access secured to any portion of a duct that segment *is carefully freed to insert several stay sutures* At this time cholangiogram is done using a needle puncture or the small incision through which an ureteral (or polyethylene) catheter is threaded (fig 73) For the cholangiogram, double strength contrast medium is used (70 per cent Diodrast or 50 per cent Urokon) Cholangiography may give accurate information concerning *location, direction and contours of the ducts* as well as evidence regarding calculi, stricture or tumor This information saves time and also may aid in arriving at a decision for definitive therapy Because of the clarity necessary for adequate interpreation the cholangiogram may have to be repeated

As soon as possible dissection of the ducts is completed and attention is paid to the site of disease When indicated, duodenotomy is done Prior to this, however *an ureteral catheter is threaded as far as possible and fluid is* injected so that if the duct be patent the papilla may be located Duodenotomy is done in the majority of cases which require reoperation on the biliary ducts

When increased exposure of the extraduodenal biliary ducts is required, only one portion of the duct circumference is dissected Tissue planes are utilized There is usually a sharp contrast between the reflections of the *normal hepatic pedicle and the thick cartilaginous tissues* which have formed secondary to serum and bile seepage associated with previous surgery Bleeding is less and dissection is simplified if the surgeon stays within the fascial structures Only when the site of disease is reached is that area of the duct dissected around its entire circumference—if it be indicated However the duct close to the liver should not be dissected from surrounding tissues for a distance greater than 1 cm in order to avoid damage to the portal vein and hepatic artery branches

In residual choledocholithiasis much dissection of the duct is unnecessary *If more than one incision into the duct is made* a distance of 1 or 2 cm should separate the e (Trauma infection, edema and dissection may alter the usual marginal anastomoses in *duct blood supply*)

3 Definitive Procedures

There are three decisions which may be made First no further surgery being contemplated the common duct is intubated and the abdomen closed Second, disease is to be eliminated and a reconstructive procedure accomplished Third, if the disease cannot be removed a by pass procedure may be done *to permit bile flow into the intestinal tract*

The first decision is made in the patient who is not in good condition and who has complete biliary duct obstruction. The duct is intubated and a large volume of bile escapes under tremendous pressure or the bile passages have been located and no bile or a clear mucus (white bile) is seen. Both conditions may prognosticate fatal hepatic failure. Decompression of a large volume of bile frequently precipitates shock by reason of blood lost from circulating volume into the liver. Precaution is to be taken for slow decompression of the ducts (Ravdin and Frazier). In the case of the acholia (white bile) hepatic insufficiency is already present.

Under such circumstances, only one other maneuver may be attempted. This is to establish a jejunostomy (or an ileostomy) for purpose of refeeding bile which drains through the indwelling tube. Having completed the bile duct intubation, with or without the enterostomy, nothing further should be done until the patient's general condition improves. Careful decompression of the bile ducts will so permit it.

A second decision may be to remove disease and to reconstruct the bile passages when favorable conditions exist (chapter 12). Procedures elected at this time may include excision of stricture and anastomosis of the duct, removal of calculi supplemented by supra- or transduodenal sphincterotomy, or reimplantation of the duct. Resection for malignancy can be accomplished (chapter 13) if indicated. Frozen section examination of lymph node or suspicious tissue may be valuable.

The third decision, to bypass an irremediable state, may be accomplished by duct-intestinal anastomosis of many types (chapters 12 and 13).

Sutures are placed at one side in the duct surrounding site of emergence of the "T" drain. The other side of the intact duct is opposed directly to the limb of the tube or drain (fig. 73b). Healing is improved, removal of the tube disrupts only one portion of the duct, stricture formation is mitigated.

Removal of a "T" tube or drain should be done only if the lateral limb of the tube can remain shut for at least 48 to 72 hours without having produced symptoms, if the studies of the duct's pressure and volumes reveal normalcy, and if cholangiography is normal. These three factors should agree (chapter 15).

Healing of the common duct following reoperation is fraught with the same disasters which produced the cause for repeated surgery. It is essential to observe, examine, identify and eliminate these causes at reoperation.

The first objective of surgery for recurrent biliary tract disorder is to maintain adequate flow of liver bile. The second objective is to provide an adequate route for bile flow into the intestinal tract. The first objective is essential to life, the second to comfort.

4 Gastric Surgery and the Bile Ducts (*Reoperation*)

Two important coincidences are common *in surgical experiences today*. One is that the patient who had gastrectomy for peptic ulcer may require cholecystectomy for cholelithiasis. The second is that the patient who had a previous cholecystectomy may need gastric surgery. *Both gastric and biliary tract surgery may be indicated in the patient who has chronic* (surgical) duodenal ulcer and cholelithiasis (with empyema).

It is preferred that each operation be done *at a separate stage*. *It is preferred*, further, that cholecystostomy (with evacuation of the calculi) and subtotal resection with gastroenterostomy (or gastroduodenostomy) be done if peptic ulcer disease symptoms have been maximal. *It is felt that if* cholecystectomy must be done that gastric surgery be postponed. I have done both operations simultaneously, *this is not to be considered a "technical triumph."* It is not justifiable to do too much in the patient with benign disease who may have hepatic damage. The situation is entirely different when dealing with malignancy.

At gastrectomy after cholecystectomy, the course of the common duct and the hepato duodenal ligament are carefully examined. The presence of many *postoperative adhesions and the existence of a penetrating post* bulbar duodenal ulcer together make it mandatory that the common bile duct be identified and intubated and the papilla be located. This may prevent *scalpel or suture damage to the duct. This duct should be drained* just as is done following choledochotomy.

At cholecystectomy following gastrectomy there usually is a deformity *of the first portion of the duodenum with abnormal geography*. As a result, the common bile duct and the cystic and hepatic pedicles are farther toward the liver and may be more posterior and lateral than normal. In such procedures adequate mobilization of the descending portion of the duodenum is mandatory in order to provide adequate exposure of the hepatic pedicle and also to place *the biliary ducts under sufficient tension to permit* careful and accurate dissection. Tension on the duodenal stump and loop is required to avoid damage to the relaxed or tented common duct.

D Exploration and Intubation of the Common Bile Duct

Even with maximal exposure and suitable anatomical conditions, exploration of the common bile duct is a *blind and hazardous procedure*. To gain full access for direct inspection of the entire common bile duct could destroy the duct and its function. Even if it can be accurately reconstructed, severe *symptomatic sequelae could appear*. The papilla of Vater is not readily accessible and unfortunately is frequently involved in complicated bile duct disease.

We depend upon indirect evidence to a great extent inspection to determine size and character, palpation with the aid of an indwelling guide measurement of pressure volume and emptying capacities, and cholangiography Common duct exploration should include these and such additional procedures as may apply in each case These are to be done systematically carefully and with due regard to friability of tissues

1 Inspection and Palpation

The fingers of the left hand are in the foramen of Winslow the thumb (and if applicable the fingers of the right hand) palpates the supraduodenal portion of bile duct Pressure is very slight examination is directed to the lateral aspect of the hepatic pedicle The thumb is abducted the fingers moved medially to the foramen of Winslow the posterior gastric wall and the head and uncinate process of the pancreas The thumb then encircles the descending limb of the duodenum and the hand caresses the head of the pancreas together with the retroduodenal and pancreatic portions of the common bile duct Observation is directed toward nodules irregularities abnormal contour or masses areas of softening

2 Duct Is Opened

Note size and character of periductal tissues diameter of duct width of wall and size of lumen color and sheen of the mucosa course the duct takes in the hepatic pedicle character of bile flow estimated or measured volume color and viscidity and the presence of debris or calculi

3 Catheterization

Place a small wet umbilical cord tape to obstruct flow toward the duodenum Pass a small urethral ureteral or polyethylene catheter towards the liver Palpate the duct containing the catheter Inject warm saline or 1 per cent novocain as the catheter is moved to and fro meanwhile inspecting the returns from lavage Remove the catheter Remove the tape and place it to obstruct the hepatic side of the duct Introduce the catheter towards the duodenum palpate irrigate Should no debris or calculi be present the occluding tape is removed and the duct irrigated again

4 Test for Flow

Introduce suitable T drain or catheter Approximate stay sutures to seal the duct Attach water manometer and three way stopcock Introduce 8 to 10 cc of saline solution Measure the pressure within the duct and determine whether the duct is emptying Adjust sutures or tube if (excessive) leakage occurs Adjust quantity of fluid introduced in the duct and repeat test to determine that pressures greater than 20 cm of water

or volume greater than 15 cc are not present and that the duct empties at the rate of at least 1 cc per minute (This test is not suitable, although it can be done through a cholecystostomy tube) Injection of colored fluorescent or radio-opaque fluids, *per se*, cannot be acceptable as evidence of normal patency to the bile duct or its pars intestinalis Since, in benign states, the papilla may be temporarily occluded by physical (cold) chemical (iodine) or neurogenic (anesthesia) factors the failure of a fluid to pass from the choledochus into the duodenum on one or several tests may not be significant In addition, abnormal states, such as the presence of calculi within diverticula or papillomata within the papilla may not obstruct bile flow

Contributory evidence concerning flow characteristics should be sought Cholangiographic evidence, manometric determinations and rates of flow palpation and careful specific evaluation (*vide infra*) are essential to confirm any presumption that the duct orifice is abnormal In many individual cases, it will be safer to consider any existing "doubt" as evidence towards an abnormality, *until adequate evidence of a normal state is obtained*

In the event of no abnormalities, a "T" drain or tube is sutured in place and cholangiogram is done (chapter 8)

If calculi are removed repeated irrigations and cholangiograms are done until the duct is clear Should the calculi be small and the termination of the duct as seen on cholangiogram normally narrow a sound probe or *dilator may be passed*

5 Instrumentation

The *instrumentation should not be done without previous knowledge* (by palpation and x ray) as to the course of the duct and its relation to adjacent structures A blunt flexible instrument in different sizes between *1 and 6 mm in diameter may be used* A slightly convex arc is prepared in the instrument It is tested for "give" against the liver no greater pressure is applied by the tip of the index finger than that which will make a slight *dimple in the liver* The operator's left hand is around the hepatic pedicle and descending duodenum The instrument is handled as a quill pen as it passes into the duct The only pressure applied to the instrument is that *of a light tap of the index finger* (If a retractor has been handled previously at least five minutes should elapse before this maneuver is done) Passage of the instrument into *the duodenum will be noted as a slight give which occurs after the tissues have appeared to grasp the instrument* The maneuver may be repeated up to a size no greater than 6 mm in diameter Should passage of the probe into the duodenum not be achieved satisfactorily under this procedure a transduodenal sphincterotomy can be done

Sounds or dilators are passed through the papilla only if there is reason

to assure an enlarged transpapillary orifice, as in the presence of small stones with recurrent jaundice When dilatation is unsatisfactory, transduodenal papillotomy should be done

Rounded stone forceps ureteral calculus procurers or any equivalent method for actual dislodgement and removal of calculi are acceptable for careful use If the duct is large enough, digital exploration is feasible Small malleable scoops (pituitary scoop) may be introduced to pick up small stones Ureteral stone grasping instruments are efficient

In patients with intrahepatic calculi an ample passage through the papilla must be assured by a transduodenal sphincterotomy or by a side to side choledochoduodenotomy The first procedure is preferred to the second since the lateral anastomoses tends to close as well as to leak

In certain selected cases of hepatic calculi, and before cholangiography[1] is done a small gauze plug is tied to a suture and inserted to occlude the distal common duct A solution of fibrin (4 to 5 cc) is introduced into the T drain or tube into the common duct followed within a few seconds (from another syringe) by injection of thrombin (300 units in about 2 cc) The duct and its contents are gently agitated by the fingers The fluids remain in contact for at least ten minutes Removal of the T drain will then bring a fibrin cast of the duct including debris sand and calculi (Fig 74) Any residual fibrin is lysed by bile after four hours and (totally) within 24 hours (Sterling 1955)

6 Comment

Prior to 1900 common duct surgery was rare When any procedure on the duct was accomplished the postoperative course was attended with more than 75 per cent mortality in the best hands The problems of hepatic insufficiency and jaundice were greater at the time than those attending primitive methods of drainage for the duct The first methods of drainage used were to place a Penrose tube drain near the involved area

Improvements in methods of procedure and management induced A W M Robson in 1902 to use double intubation with catheters J B Deaver in 1904 to utilize a T' tube and Kehr in 1909 to accept the use of the T tube (or Kehr drain) as a routine procedure in postoperative management of the common bile duct

There are some today who disdain to use a drain or tube in patients who have had common bile duct exploration The majority opinion (well over 95 per cent) is that intubation of the common bile duct following choledochotomy provides a safety valve for the biliary tract A patient who had even partial use of the gallbladder prior to choledochostomy at the time

[1] Diodrast produces floccules in contact with fibrin and thrombin and delays clot formation



FIG 74 FIBRIN CLOTS (FORMED BY ADDITION OF THROMBIN TO FIBRINOGEN) IN A BILE DUCT ENMESH BILIARY SAND DEBRIS AND CALCULI

This clo e up photograph of such clots demon trates whole and fragmented cal culi attached to clot s edges adherent to attenuated portions of it and al o pre ent at the tip of the clot

of cholecystectomy is capable of withstanding up to 20 cm of bile pressure within the duct That degree of pressure may be required to open the nor mal papilla of Vater As a result the duct incision may leak and a symptomatic subhepatic abscess can form This is seen often following needle cholangiography When the external limb of a T" tube is clamped during the first three to four days postoperatively, the patient usually has a pres sure in the epigastrium occasionally with severe pain and generally ac companied by leakage of bile around the "T tube

In addition, no matter how carefully the common duct exploration is done at surgery there is always more information which can be obtained by postoperative clinical and pressure volume studies cholangiography and laboratory examination of the excreted bile

The tube in the duct permits full freedom from complications for years even in the presence of choledocholithiasis dyskinesia of the papilla stric ture of the duct and cholangitis The value of an indwelling tube in the duct is so great for the few patients who need it that denial of its pos ible u e to every patient is a risk

Therapy such as is utilized in the conservative management of residual choledocholithia is is feasible in certain cases through the "T tube

Further value is evidenced by the strut function performed by the indwelling drain in prohibiting stricture formation along a lengthy longitudinal incision in the duct In patients with end to end or other types of common duct anastomosis the indwelling tube protects the anastomosis and permits adequate flow across it

The "T" tube in common use today is made in a variety of widths from French size 12 through 24 The 'T' is about four inches long, the lateral limb may be from 12 to 16 inches long The "T" is cut to size for individual use

This "T" tube provides a complete circle through which bile drains The fit between tube and duct is usually snug so that very little if any bile goes around the tube This may be advantageous in permitting rapid healing of the duct wall in end to-end anastomoses Modifications have been introduced such as a double lumen for later irrigation a long limb to the "T" to be passed through the papilla an attached Foley bag to prohibit inadvertent loss of the "T" tube tabs attached to the lateral limb to permit suture attachment of the tube to the skin and a "Y" extension on one of the "T" limbs for insertion into the hepatic ducts

I have not been satisfied with the complete circle of the "T" tube First the junction between the vertical and horizontal limbs frequently has to be checked for patency this means a cut has to be made into the tube Second

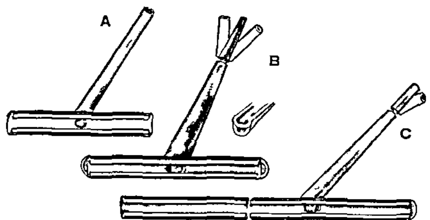


Fig 75 T Drains

A A canal rather than a tube is used as the horizontal limb within the bile duct
B The (canal) "T" drain has supplementary return flow lumens intended for irrigation and cholangiography with benefit of optional directional control
C Cattell type long limbed "T" drain with a canal rather than a tube inserted into the bile duct and through the papilla into the duodenum A return flow channel is incorporated with the short limb which is placed toward the hepatic radicles (Courtesy of *Surg Gynec & Obst*)

the horizontal limbs of the "T" have to be cut and trimmed to fit without angulation or excessive pressure on the duct walls This cut may occlude the tube lumen There is also a tendency for the irregular edges of the tube to catch debris

I prefer a "T" drain (fig 75) wherein the tube is converted to a half circle In the short limbed "T" drain a double lumen can be used for each limb of the "T" Irrigation of the duct then is no longer a "to fro" motion but becomes a continuous stream directed to force any retained debris or small calculi through the larger lumen

Furthermore, the "T" drain has been adopted with the addition of a long limb on one of the "T" 's This length may be cut as desired at the time of (transduodenal) sphincterotomy In this type of "T" drain, a doubled lumen is placed on the short limb of the "T" in order to permit irrigation of the hepatic radicles as needed and also to permit adequate cholangiography The doubled lumen into the duodenum permits feeding (Rhoad) and occasionally intestinal decompression

Many improvisations have been adopted for postoperative care of the "T" drain or tube (chapter 15) For example plastic tubes have been attached or the tube drains through a nipple into a bottle attached on the scultetus binder or into the barrel of a syringe, the nozzle of which drains through a long tube

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provide free flow to bile and to identify the cellularity of the tissue at the papilla

Blind procedures such as external approach to the sphincter (Gillette has used a Ramstedt Fredet procedure for sphincterotomy) and needling the papilla or pancreas have been used These are not advantageous

Combined supra and transduodenal exploration of the common bile duct may be indicated on one third of patients who require choledochostomy

a *Mobilize descending duodenum by incision (Kocher) of lateral leaf*let of the peritoneum (fig 71)

b Pass catheter or soft probe into area of papilla (fig 73) from the previous choledochotomy incision

c Locate papilla by palpation over intracholedochal probe and by injection of fluid *Place traction sutures to outline proposed longitudinal* incision into antimesenteric aspect of lower descending duodenum (fig 76)

d Incise duodenal wall Obtain accurate hemostasis Identify papilla and place traction suture into inferior aspect of apex (fig 77) Pressure on a probe passed through the supraduodenal bile duct may advance the papilla into the area of the duodenotomy in certain patients Lateral edges of the duodenal opening may then be depressed around

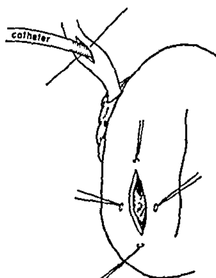


FIG 76 SPHINCTEROTOMY—I

Four traction sutures are placed into the wall of the mobilized duodenum Longitudinal incision is made along peritoneal attachment of descending limb of duodenum A catheter or probe has previously indicated the approximate location of the papilla A cholangiogram has been done This vertical incision in the duodenum will be closed transversely later (cf Fig 81)

the advanced papilla In most patients this procedure is not feasible however and finger retractors (or stay sutures) are used to separate the duodenotomy incision for exposure of the papilla

e Insert blade of scissors into lateral aspect of common bile duct orifice and continue incision through duodenal mucosa push away duodenal wall and continue scissors cut into papilla (fig 78a)

f Supraduodenal probe or catheter is advanced through the papilla

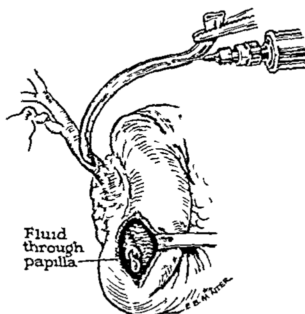


FIG 77 SPHINCTEROTOMY—II

Duodenotomy opening is retracted while fluid is injected through a catheter in the common bile duct

under vision Incision through papillary sphincter provides lumen of at least 6 mm (fig 78b)

g Catheter (urethral) or long limb of T tube or drain is passed through papilla the vertical limb emerges through the opening in the supra duodenal common bile duct (fig 78c)

It may be difficult to locate the papilla In about half the cases the papilla will be elevated above duodenal mucosa and palpable as well as visible In the other half it may be depressed although still palpable In about 1 per cent of patients it is present in a congenital diverticulum of the duodenum

The tube (or drain) through the papilla should be of sufficient length that it cannot be easily dislodged and short enough to avoid knots Usually

four to six inches of the transpapillary tube should extend into the duodenum

B Benign Disease of the Papilla

Abnormalities of the papillary (or transduodenal) segment of the common bile duct are often associated with pancreatitis and *biliary dyskinesia*

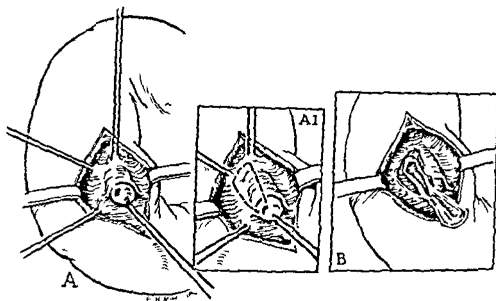


FIG 78 SPHINCTEROTOMY—III

A Three or four traction sutures are inserted for control of the papilla Sutures are not placed medially

A *Incisions into duodenal mucosa and papilla are made at 9 o clock for simple sphincterotomy or at 9 and 12 o clock for removal of tissue to be examined microscopically (the latter procedure is preferred)*

B Flap is elevated to expose bile duct termination The base of the flap is the *apex of the papilla*

Regardless of theory or cause abnormal interductal reflux which is associated with symptoms and disease should be remedied

Traumatic interductal fistula is found in the proximal segment of the papilla when associated with *pancreatitis Incision in the terminal half of* the papilla will *not* be effective in overcoming *sphincter action* Plastic repair of the sphincter *requires incision of the entire sphincter* Free flow from the common bile duct is thereby assured

Care is essential to avoid damaging the pancreatic duct Sutures are placed laterally in the papilla (fig 79) and then only with a probe in the

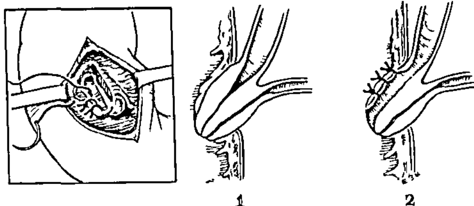


FIG 79 SPHINCTEROTOMY—IV

A Sutures are placed to approximate the duodenal mucosa and bile duct

B A diagram illustrates 1) the intact sphincter and 2) the appearance of the bile duct in the papilla after sphincterotomy

pancreatic duct In some cases of pancreatic duct obstruction it may be necessary to revise the pancreatic duct orifice in order to guarantee free flow from it Accordingly sutures are placed only medially between the duct and papilla in order to avoid including any portion of the common bile duct

1 *Sphincteroplasty*

Sphincteroplasty includes repair to the ducts following

a Excision of wedge of papilla for biopsy

b Excision of lateral wall of papilla to establish free flow from the extra duodenal common bile duct

c Excision of medial wall of papilla to assure free flow from pancreatic duct into the duodenum

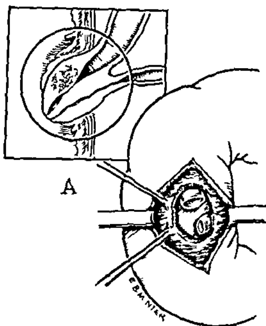
Repair is accomplished by suture of duct mucosa to the duodenal wall medially (in pancreatic duct plasty) or to the lateral duodenal wall (for common bile duct repair) (fig 79) Indwelling tubes pass through the ducts into the duodenum The tube in the pancreatic duct emerges through a duodenotomy and a separate stab wound or it may be passed in retro grade fashion to emerge through the body or tail of the pancreas (caudal pancreatostomy) Short catheters or tubes may be secured by a suture the long end of which is cut after 2 weeks thereby permitting intestinal peristalsis to carry the tube down

Sphincteroplasty should be done to supplement and protect repair to com mon bile duct stricture In addition following excision of bile fistula or duct stricture Dragstedt has suggested use of the transduodenal approach to

identify the distal common bile duct for anastomosis to the common hepatic duct

2 Resection of Papilla (Fig 80)

The entire papilla may be excised to remove organic disease and other defects associated with trauma from pre existing calculi, pancreatitis, instrumentation or unsuccessful attempts at repair



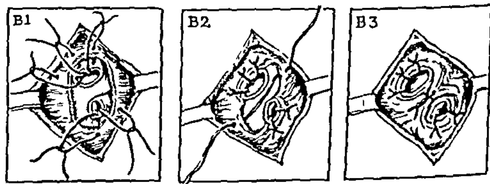


Fig 80 Excision of Papilla

A Extent of excision of the papilla is indicated by the circle The papilla together with the immediate extraduodenal segment of the ducts are to be resected

B Sutures are placed for reimplantation of the ducts between contiguous duct and duodenum walls (B1) Pancreatic and bile ducts are not sutured to each other Opposite duodenal walls are approximated with one or more mattress sutures (B2) Following completion of the reimplantations (B3) indwelling tubes may be placed

One technique of excision of the papilla is as follows (fig 80)

a Duodenal mucosa is incised in a circle surrounding the papilla
b Mucosa and submucosa are dissected away from the papilla The extraduodenal common bile and pancreatic ducts are identified Stay sutures are placed in each duct
c The papilla is amputated after cutting through ducts
d Sutures approximate the lateral aspects of the common and pancreatic ducts to the adjacent full thickness of duodenal wall (fig 69)
e A long limbed 'T' tube (or drain) is passed through bile duct The external limb of the 'T' emerges through extraduodenal choledochostomy A short segment of urethral catheter is retained by a chromic catgut suture in the pancreatic duct

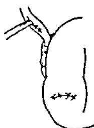


FIG 81 DUODENOTOMY CLOSURE

Original longitudinal incision (cf fig 76) is closed by bringing ends of incision together in a transverse direction The closure is reinforced by omental graft (not shown)

f The space between the pancreatic and common bile ducts has no duodenal wall If gap greater than 1 cm is present duodenal wall can be closed with interrupted sutures between the ducts
g Fine catgut or silk sutures may be used to approximate mucosa of common bile or pancreatic duct to adjacent duodenum
h The duodenotomy is closed transversely to avoid stenosis (fig 81) Free omental transplant may be utilized for reinforcement
i Duodenum is replaced and may be sutured to the parietal peritoneum The duodenotomy area should be drained by tube or sump
j T drain is sutured in the extraduodenal common bile duct and fixed to fascia subcutaneous tissue or skin

C Stricture of the Extraduodenal Bile Ducts

Sir James Walton wrote in 1944 that every removal of a gallbladder is fraught with very great danger and one small error may convert a patient with a mild disability into one in immediate danger of death faced with

prolonged misery and many subsequent operations which, at best, may only give an incomplete life."

Thus, in gently ominous language, is stated the occasion wherein a common bile duct stricture *may be produced in 75 to 80 per cent of cases* This cannot fully describe the apalling situation which develops as the cystic artery begins to pump away from its ligature Nor can this state the full sense of calamity which is approached *after cutting the cystic duct to find* that it actually was the common hepatic duct which was severed Nor has *any prose yet been written to portray* the surgical anguish which accompanies increasing jaundice after a simple cholecystectomy

The misery, calamity or anguish represent a full conscience However, the error found as anatomical anomaly has its counterpart in the error which may be made by the most talented, most careful and most meticulous surgeon *The qualified surgeon* can appreciate the error, can extricate himself and can remedy the defect It should be clearly understood that there is no place for reprimand guilt, intolerance nor abysmal defeatism in such a situation except *to a minor and transient degree* It is most important to recognize the problem and intelligently, to solve it

The best time to repair damage to the bile ducts is at the original operation Should a technical deficiency be observed in the immediate postoperative period, a retained stone may be removed after two or three weeks an obstruction relieved between four and six weeks and a bile fistula corrected immediately or after three months

Trauma at surgery is not the only cause for choledochal strictures There are congenital forms and others due to inflammation external to the duct collections of bile and fibrosing pancreatitis which can also cause stricture of the extraduodenal duct Stricture at the papilla is commonly caused by trauma to or inflammation of the sphincter

Traumatic stricture is usually located in the common hepatic duct or at the junction of hepatic and common bile ducts Inflammatory and other types of stricture usually involve the common bile duct *per se*, between the junction of cystic and common bile ducts to the level of the papilla Stricture due to malignancy may occur at any site In order of frequency *stricture occurs at the extraduodenal* common bile duct, hepatic duct cystic hepatic duct junction and papilla

The presence of a stricture may be suspected in the presence of a persistent bile fistula It may be suspected in a patient who has progressive obstructive jaundice *following cholecystectomy In the patient who has a* choledochostomy cholangiography will be illuminating

Common bile duct stricture may not be recognized after surgery *The duct lumen may be narrowed* yet if liver function be normal and the

papillary sphincter be patent, the stricture can be asymptomatic and harmless

Jaundice is observed in 87 per cent of patients with complete stricture The other 13 per cent have a bile fistula In patients who have a bile fistula together with duct stricture jaundice is observed in 50 to 55 per cent

1 Excision with End to End Anastomosis

The ideal treatment of stricture is its excision with restoration in continuity of the bile duct by end to-end anastomosis This method applies without the need for excision of damaged tissues to the immediate repair which is utilized if injury is noted at the time of cholecystectomy Should the common duct laceration *involve not more than 50 per cent of the* circumference the defect can be approximated over an indwelling "T" tube or drain The vertical limb of the "T" is withdrawn through another site

Excision and end to end anastomosis (mid portion of common bile duct)

a The site of stricture is cleared by sharp dissection It is approached from normal duct above and below using only one aspect of the circumference of the normal duct to approach the area of stricture The site of stricture and approximately 1 cm of the total circumference of the uninvolved duct are freed

b Traction or stay sutures are placed in the normal ends of the duct

c Hepatic artery and portal vein are identified

d The duodenal reflection has already been incised and the descending limb of the duodenum together with the head of the pancreas mobilized This mobilization is improved if indicated

e Three through and through silk sutures (000) are placed between proximal and distal duct, these are not tied (fig 82a)

f A cobbler s (or baseball) stitch is placed to approximate the posterior wall (fig 82c)

g The vertical limb of a 'T' drain is passed either proximally or distally through a small opening made in the duct at least 1 cm away from the anastomosis It is cut to fit smoothly without any irregularities and without angulation (fig 82b)

h The anterior wall is completed by flat mattress sutures through all layers Eversion is not harmful except for its tendency to permit leakage Excess inversion may form a septum in the duct (fig 82d)

i Stay sutures are removed Fascial layers surrounding the ducts may be approximated

j The superior lateral aspect of the duodenal loop is sutured to the lateral edge of the hepatoduodenal ligament or to the liver to avoid tension at the anastomosis

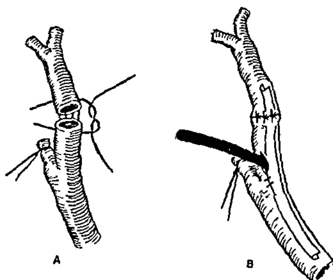


FIG 82 REPAIR OF COMMON BILE DUCT

A End to end anastomosis is accomplished by a single row of sutures This is reinforced at the angles to secure very slight inversion of the mucosa

B Choledochostomy for "T" drain splint is established distal (in this case) to site of bile duct anastomosis

In accomplishing bile duct anastomosis it is important to

1 Avoid tension
2 Remove diseased tissue
3 Approximate mucosa
4 Use a "T" drain through the anastomosis with a vertical limb which emerges elsewhere
5 Assure free flow through the papilla
6 Exclude other duct disease
7 Permit enough time for adequate healing

2 Excision of Stricture (at Junction of Cystic and Bile Ducts) with Replacement by Prosthesis or Bowel

Various materials may be substituted for a defect in the bile ducts Some methods are ingenious and amazing In all cases transplanted tissue usually disappears occludes or structures again In addition a bile or intestinal fistula may be present

It should be emphasized that the use of prosthesis and tissue substitutes in the bile duct are not satisfactory It is preferred that the proximal end of the duct be reimplanted or be anastomosed to some segment of the intestinal tract

Autogenous tissue used as substitute for the common duct includes vein

defunctionalized intestinal limb (with or without mucosa) peritoneum fascia uterine horn and tube ureter skin (as a lined graft pedicle as split thickness roll or as cutis graft covering or insert) Banked tissue such as fascia and blood vessels have been used Substances such as vitallium stainless steel tantalum monel metal bouncing clay fiber glass polyethylene polyvinyl and other plastics have been reported Rubber tubing has remained the most satisfactory splint

The disadvantages of the rubber prosthesis are its tissue irritation its low tensile strength which requires a thick wall in relation to the size of the lumen its tendency to become brittle to disintegrate and fragment

Vitallium has the advantage that it has a relatively thin wall with a large caliber lumen there is very little tissue reaction to it It can be molded welded and custom made and has minimal irregularities to act as a nidus for conglutination of debris On the other hand vitallium has to be removed surgically It can become plugged There are also many instances of abscess formation Many workers (Grindley Palomba) are very enthusiastic about polyethylene Because it is planned that this be used for a short term there probably is no problem of the carcinogenesis usually met in the synthetic hydrocarbons (Oppenheimer et al)

Prosthetic appliances furnish good material for investigation However nothing will substitute for proper use of the patient s tissues carefully approximated without tension with accurate technique and with clear understanding of the fundamental principles involved

The use of a prosthesis or the substitution of a segment of bowel between two separated ends of bile duct means twice the danger of leakage and double the tendency to future stricture formation When a large gap exists which cannot be overcome by mobilization of the duodenum then it is preferred to re implant the proximal duct If duct to-duct anastomosis cannot be done without tension duct to-intestine anastomosis is next preferred

Procedure for insertion of prosthesis

a Expose one aspect of the entire common bile duct at stricture and at least 1 cm on either side

b Mobilize duodenum and free hepatic pedicle to bring distal segment toward liver hilum

c Excise area of stricture

d Insert prosthetic appliance do not tie around the cuff Attach duct and tube to adjacent tissues using wire or other non absorbable suture Bring edges of gastrohepatic ligament around the tube and junctions with ducts

e Make additional opening into available site at either side of anastomosis thread catheter or T drain through it

f Transcholedochal or transduodenal sphincterotomy should be done and the indwelling catheter passed through into duodenum if desired

3 Duct to Stomach Anastomosis (Tube Pedicle)

The blood supply of the stomach and duodenum is sufficient to form a full thickness flap of the wall and to construct a tube

Moynihan has used trap door flap of stomach Walton cut a flap with base from the side of the duodenum which was used for the newly constructed tube

a *Steps (a) through (c) in section C 2 above*

b Close distal common duct stump (Parker Kerr)

c Prepare tube originating at the greater curvature of the stomach with its *base on the lesser curvature Establish "Spivack" type of valve if desired (fig 83a)*

d Close defect in stomach

e *Place "T" drain to emerge through bile duct proximal to the distal* cut end horizontal limb may cross entire gastric tube to the stomach Urethral catheter may be used

f Mucosa approximation *between bile duct and stomach tube (fig 83b)*

g Serosa of stomach tube attached to periductal tissue, cuff formed

h Additional row of serosa periductal sutures placed to overlap the anastomosis and *to* avoid tension

i A flap of omentum is sutured around the anastomosis

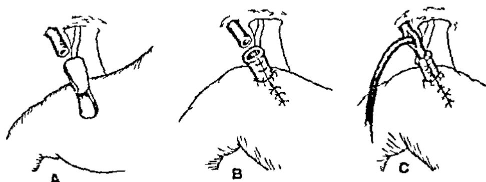


Fig 83 Intestinal Tube Anastomosis to Bile Duct

A Full thickness flap is elevated from stomach or duodenum This is placed as demanded by individual anatomical characteristics

B Tube is fashioned from flap by two layers of interrupted sutures in mucosal and serosal layers

C Anastomosis between end of bile duct and end of gastric (or duodenal) tube is completed over an indwelling "T" (tube or) drain the external limb of which emerges through a fresh opening in the bile duct

Procedure for use of pedicled small bowel segment

a Segment of jejunum is isolated including its blood supply, intestinal continuity restored by end to end anastomosis (mucosa may be stripped)

b Vascularized intestine brought through incision in mesocolon (right side)

c Two end to end anastomoses done between bowel and ducts (interrupted sutures)

d Catheter inserted through incision in intestine or duct for decompression purpose

D Fistula

A bile fistula tends to close It will remain open under two conditions 1) if there be obstruction to bile flow or 2) if the walls of the common duct are infected

Lateral (postoperative) biliary fistula should close within three to six weeks Partial biliary fistulae may persist for years

Choledochitis or a pericholangitis may be observed at operation when the wall of the common duct is thickened when there are enlarged lymphatic glands and when there is hyperemia and congestion of the duct mucosa and periductal tissues In such states prolonged drainage of the common bile duct is indicated (chapter 15) Antibiotics are used depending upon sensitivity spectra

Bile fistula develops most often in patients who have 1) serious inflammation including choledochitis and cholangitis, 2) multiple intraductal calculi producing longstanding trauma to extraduodenal duct wall 3) severe trauma to the papilla produced by manipulation or calculi 4) poor technique in insertion of intraductal drain or if the tube or duct be disproportionately large or if sutures have been placed to involve both walls of the duct and 5) inadvertent technical error during which the common duct has been opened on both sides

1 Transplantation of Fistula

Williams and Smithwick (1929) successfully implanted an external biliary fistula to the duodenum This procedure however is used only as a last resort

When the common duct cannot be found a fistula may be purposely formed Dissection is carried into the porta hepatis until a place is found from which bile drains A catheter is then sutured into this region and brought externally Three or four months are allowed to pass for the fistulous tract to form and to establish a good blood supply

In the transplantation dissection is in the form of a cone, leaving the base of the fistula (the part nearest the liver) wider than the end to be used in the anastomosis No tension should exist between the bowel and base of the fistula Adequate circulation in the fistulous tract is essential for the success of this procedure

There are two defects in this First, connective and fatty tissues in the tract often can contract One way to avoid this is to use a replaceable tube indefinitely The second defect is that any duodenal opening is subject to leakage as well as subsequent stenosis

Wilson advocated making a serous lined tube from the stomach to incorporate the fistula, and Dragstedt et al (1943) advocated the use of the duodenal wall Fascia lata, pleura and peritoneum have been used

The operations of Wilms (1912) and Sullivan, both of whom turned an omental graft about a tube, have not been successful When an epithelial lined tube or a direct anastomosis cannot be established the patient will have biliary colic, cholangitis and hepatic insufficiency

2 End to Side, Duct to Duodenum (Fig 81)

In the absence of normal function to the papilla there may be severe cholangitis and recurrent stone formation However, if bile flow is free and constant and if there is no obstruction to bile flow, then the incidence of infection of the biliary duct following duct duodenal anastomosis is minimal W J Mayo first described the method used today for accurate anastomosis between duct and duodenum Anastomosis in this situation can also be accomplished by use of an intestinal wall flap which is sewn around the common duct as a nipple

Choledochoduodenostomy is done by approximating the mucosa of the common duct to the mucosa and submucosa of the bowel with interrupted sutures The outer intestinal wall is anastomosed to the outer wall of the common bile duct by interrupted silk sutures Too much pleating of either the common duct or the intestine is avoided

In this type of anastomosis a 'T' tube is passed from the proximal portion of the common bile duct through the site of anastomosis The anastomosis is reinforced by an overlapping of a portion of intestine pulled up over the site of anastomosis

3 End-to Side, Duct to Duodenum (Coffey I, Modification)

For many years ingenious general surgeons have adapted urologic technique in repair of the common bile duct I observed Deaver and Behrend using modifications of the Coffey I (ureteral intestinal anastomosis) procedure for choledochal-duodenal anastomoses

This is indicated in patients with traumatic stricture or fistula of the

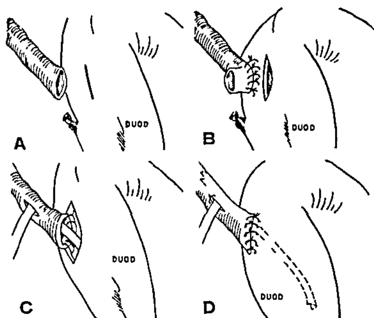


FIG 84 CHOLEDOCHODUODENOTOMY (END TO SIDE)

A Distal bile duct is ligated Incision into duodenal wall is placed posterior to the peritoneal reflection

B Interrupted sutures are placed between the postero lateral duct wall (proximal) and duodenum

C *Long limbed T drain is passed through the anastomosis* The vertical limb of the T drain emerges through a fresh incision in the bile duct An inner row of sutures is placed

D Anastomosis is completed Duodenum is buttressed against the hepatic pedicle to avoid tension on the anastomosis

duct wherein mobilization of the duodenum is prohibited and when excessive adhesions preclude use of a long loop or limb of jejunum for anastomosis

The common bile duct is prepared as for end to side anastomosis (end of duct to side of duodenum) It is cut obliquely and a small longitudinal slit made to form a spatula tip A chromic catgut (000) suture is passed through the tip of duct wall knotted and needles mounted on both ends of the suture (fig 85a)

The duodenal wall is incised along an accessible (antimesenteric) aspect The muscular coats only are incised for about 3 cm The incision is spread by blunt dissection to expose a pouting submucosa (fig 85b) The most distal portion of the incision is then perforated (scalpel or electrocautery) (fig 85c)

Each of the needles is passed through the opening in the duodenal lumen

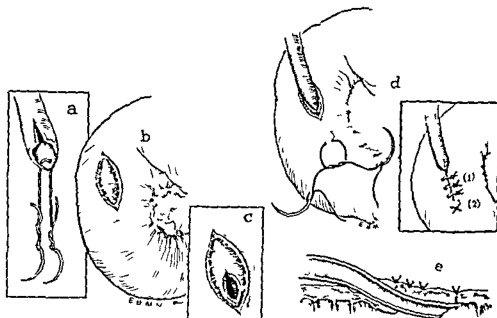


FIG 85 CHOLEDOCHODUODENOSTOMY, END TO SIDE (COFFEY I MODIFICATION)
A Preparation of the transected common bile duct doubled suture with needle at each end is inserted in the tip of the duct Longitudinal incision converts end of duct to spatula like tip
B Preparation of duodenum incision into serosa exposes the muscularis
C Small distal incision through muscularis and mucosa into duodenal canal
D Needles attached to bile duct suture are passed into distal opening and out through intact duodenum about 2 cm further distally
Serosa of duodenum approximated with interrupted sutures as a tunnel for the bile duct Tension and compression are avoided
E The course of bile duct through duodenal wall is seen on lateral section upon completion of this type of choledochoduodenostomy

to carry the tip of the common bile duct distally about 2 or 3 cm The needles are passed through the intact duodenal wall and the catgut tied (fig 85d) The opening in the duodenal muscularis is approximated with several Cushing type sutures of fine silk to form a tunnel (fig 85e) Serosal sutures are placed The anastomosis is protected by available omentum

Catheter or T drain may be passed through the duct into the duodenum if desired

4 Duct to Duodenum, Side to Side

In a chronic biliary fistula the distal extraduodenal common bile duct may be distorted or obliterated by adhesions It may be feasible to free the duodenal cap and superior portion of the descending limb However additional dissection can be hazardous because of bleeding, friability and prolonged anesthesia It may therefore be elected to anastomose the com

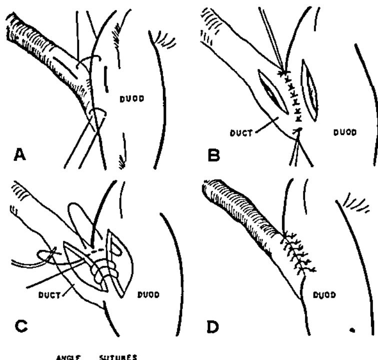


Fig 86 Choledochoduodenostomy (Side to Side)

A Incisions are made at available sites in walls of dilated bile duct and duodenum

B A posterior row of interrupted sutures is placed between the duct and duodenum

C Angle sutures are placed superiorly and inferiorly

D An anterior row of sutures is placed Completed anastomosis is reinforced by omental graft (not shown)

mon bile duct and duodenum by side to side anastomosis A duodenotomy may have already been done The papilla may not have been identified

An opening made in adjacent duodenum can be anastomosed to the common bile duct (fig 86) This anastomosis is similar to any side to side anastomosis done in the intestinal tract A drain tube or catheter is passed through the anastomosis

The duodenotomy opening is preferably made in the antimesenteric border of the duodenum close to the hepatic pedicle Two layers of interrupted silk sutures approximate mucosa and submucosa in the inner row and the outer coats of duct and duodenum in the other row Because of the active peristalsis passing through this portion of the duodenum I have used the Y T ' tube of Cattell with the Y portion (usually inserted in the hepatic ducts) placed in the duodenum as an anchor

This type of anastomosis is subject to fistulation To remedy this a sump drain is placed and a feeding jejunostomy is established

Results of lateral duct to-duodenum anastomosis are satisfactory providing that the splinting "T" tube or drain is left in situ for at least six to eight months

5 End of Duct to Loop or Limb of Jejunum

In most cases the duodenotomy for exploration of the papilla is not available for anastomosis to the duct It is usually located on the lowermost portion of the descending limb of the duodenum and anastomosis of duodenum to duct would be under tension In addition, normal flow through the duodenum may be obstructed by a twist or kink Accordingly, a long limb of jejunum is brought over or through the mesocolon for anastomosis to the common duct This procedure utilizing a long defunctionalized loop or limb of jejunum, is often used in patients who have fistulization in the hepatic ducts and in whom the distal common bile duct cannot be identified or be made useful

The Roux en Y procedure utilizing a limb of jejunum is useful for restoration of bile flow into the intestinal tract There are two disadvantages to it One is that the mobilization of the jejunum and the establishment of entero anastomosis is awkward and difficult and frequently has to be accomplished through an opening in the mesocolon Second, the probability for formation of intestinal fistula is great This latter often is followed by subhepatic abscess

Allen (1945) developed the short segment of the hepatic duct in the liver sulcus and anastomosed the open end of the transected jejunum to the liver around a tube placed in the duct He re established intestinal continuity by implanting the proximal segment of jejunum into the distal segment by the method of Roux (fig 87 and 88) This resulted in a mechanical arrangement whereby the intestinal current was directed away from the liver

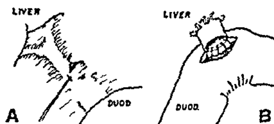


Fig 87 Hepatic Duct Anastomosis—I

A Obliterated tissue at liver hilum is separated to identify bile (duct)

B The posterior duodenal wall is sutured to the liver hilum Anastomosis is done by interrupted silk sutures Anterior aspect of duodenal wall is telescoped over anastomosis (Procedure is not usually suitable as it often produces angulation to the duodenum)

Whenever the duct is anastomosed with a lumen wider than the circumference of the duct the duct wall may be split longitudinally to increase the area used for the anastomosis

The hepatic ducts at their bifurcation may be utilized by cutting the septum between them and using the wider area for anastomosis to an intestinal lumen (Cattell) Sometimes a fulgurating loop can be used to dissect away tissue at the liver hilum until an available intrahepatic segment of duct is exposed

F Congenital Anomalies

It has only been since 1917 that progress has been made in the treatment of the congenital atresia of the common bile ducts Historically, Holmes Thompson and Ladd pioneered in the repair of congenital defects within the bile ducts The infant with obstructive jaundice does not have to be operated within the first month of life It is perfectly feasible to wait until the infant is three months of age if constitutional symptoms countenance this delay Growth is permitted in order to obtain a larger duct with which to work and in order to provide an improved risk It should be remembered that obstructive jaundice in a child is not necessarily fatal

The principles for repair of duct atresias are threefold 1) Permit the growth of the child to optimum size (for reconstruction purposes) 2) Provide diversion of the bile flow 3) Reconstruct a route for bile flow into the intestines

1 Hepatic Duct Drainage (Hepaticostomy)

Obliteration of the extrahepatic biliary ducts occurs when least expected It is commonly seen as a congenital defect It may also be a sequel to repeated attempts to repair of duct stricture and fistulae In such cases intense greenish discoloration of the liver reflects severe intrahepatic bile duct dilatation In some cases the major ducts do not enlarge Either the

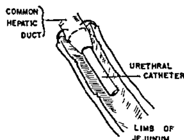


FIG 88 HEPATIC DUCT ANASTOMOSIS—II
Cored hepatic duct (at hilum) anastomosed over a catheter to jejunal limb (cf text)

smaller radicles or the dilated ducts may act as a route for bile flow Because of duct arborization, drainage of one main duct may drain most of the liver

The right anterior hepatic duct is most accessible because it is adjacent to the gallbladder bed However, the left hepatic duct may be reached immediately from the hilum Intrahepatic decompression of the liver ducts is the only feasible procedure in the patients who are very weak and in poor general condition This is particularly advantageous in patients who have *congenital atresia or longstanding obstruction*, in whom it is essential to decompress the obstructed bile flow

In several situations we have seen drainage of a large hepatic duct to permit distal edema and other obstructive phenomena to subside Normal flow through a previously damaged route was then possible Such a patient was operated in 1950 for relief of obstructive jaundice The common bile duct could not be identified because of much surrounding necrosis *Following the drainage of an intrahepatic hepatic duct, flow returned* through the common bile duct and has continued since in a perfectly well patient

In congenital atresia of the bile duct anastomosis of the gallbladder to an existing enlarged intrahepatic duct may be done if the common hepatic duct is atretic (fig 89) The right or left hepatic duct may be used Anastomosis of gallbladder to the liver will permit bile flow from one of these

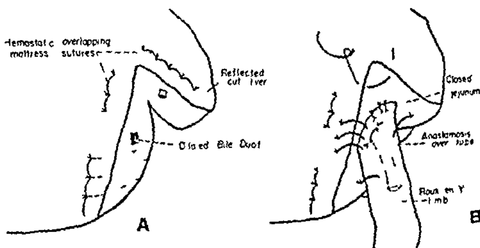


Fig 89 Hepatic Duct Anastomosis—III

A Left hepatic duct exposed after amputation of left lobe of liver (anterolateral segment is illustrated)

B Defunctionalized limb of jejunum is brought through mesocolon for anastomosis with intrahepatic duct Jejunojejunostomy completes the Roux en Y procedure

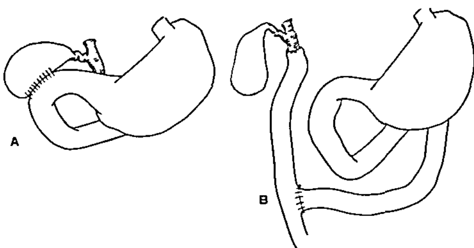


FIG 90 BILIARY TRACT BY PASS PROCEDURES
A Cholecysto duodeno tomy (side to side)
B Cholodocho jejuno tomy using defunctionalized limb of jejunum by Roux en Y procedure This anastomosis may be antecolic or transmesocolic

large hepatic ducts through the gallbladder by way of cystic duct with a normal common bile duct (fig 90)

2 Hepaticojejunostomy Side to Side

Hepatojejunostomy gives only 18 per cent satisfactory results Walters has stated that the complication of ascending infection after duct to intestine anastomosis is due to recurring stenosis rather than regurgitation of intestinal contents

Cole reports 78 per cent excellent results with use of a Roux limb of jejunum in hepatic duct anastomoses He anastomoses the Roux en Y limb to a duct located at the hilus by needle aspiration (fig 87)

Attempts to repair defects in the ducts by bridging them with tubes of rubber vitallium or other materials have poor results The tube either passes into the duodenum leaving scar tissue to contract or becomes plugged with bile

However when the extrahepatic ducts are obliterated Longmire has suggested partial amputation of the left lobe of the liver to obtain a duct for anastomosis to a jejunal loop Atrophy will occur in obstructed segments whereas the drained lobes can hypertrophy in compensation

Wilson Gillespie Longmire and Sanders and others who have demonstrated that a large dilated intrahepatic duct can be anastomosed side to side with a loop or limb of jejunum (fig 88)

There may be from two to three inches of available common bile duct

distal to the junction with the cystic duct This three inch segment may be dissected away *from the pancreas in about half of the cases* In the one half of cases in which the common bile duct cannot be freed at all the duodenum is mobilized towards the liver

"T" tubes crossing the line of anastomosis may remain in place at least six months to one year It is important that the "T" tube should be introduced through a slit above or below the line of anastomosis and that it cross the line of anastomosis to minimize contact with the bile It is also *advisable to keep the "T" tube in place so that the tube may be irrigated* free from debris

The common hepatic duct may not be identified In such cases a small section of "Y" tubing is used with a limb in each of the intrahepatic ducts When the common hepatic duct has been damaged, it is preferred to anastomose each hepatic duct separately, within the hilum Procedures to bring these hepatic ducts together for a single anastomosis are followed by *obstruction to both*

Atrophy, infection and co existing disease may make the distal end of the common bile duct difficult to find Under such conditions an external biliary fistula can be formed by hepatic duct drainage

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13

SURGICAL PROCEDURES FOR TUMORS OF THE BILIARY TRACT

A General

Tumors involving the biliary tract vary widely in symptoms pathogenesis and operability An important feature particularly at the papilla is the number of different malignant tissues which may involve the biliary tract Within a 2 cm cube at the papilla are pancreas bile duct and duodenum Malignancy arising from each has separate characteristics varies in prognosis and should be differently treated

✓Malignant tumors of the bile ducts as those elsewhere vary in accordance with location cellular characteristics and biologic potential A tumor of the papilla only a few millimeters in diameter may produce obstructive jaundice early in its history By contrast a tumor of the gallbladder may completely replace the right liver lobe and the first symptoms of anorexia pain jaundice and vomiting occur just a few weeks before death Similarly a tumor at the inferior anterior border of the pancreas may be silent until the tumor has irrevocably cemented adjacent abdominal structures

✓ Bile duct carcinoma is locally invasive through perineural and adjacent tissues Lymphatic and hematogenous metastases occur much later in their life history than with carcinoma of the duodenum or pancreas Surgical eradication may be considered unless the superior mesenteric vessels are infiltrated Liver portal vein duodenal pancreatic and renal capsule involvement need not prevent surgical extirpation Should resection not be feasible by pass procedures may be effective For example carcinoma at the papilla of Vater may be by passed happily by simultaneously doing (a) cholecystogastrostomy (or choledochojejunostomy) (b) gastrojejunostomy and (c) pancreatic duct jejunostomy

Malignant lesions involving the pancreas spread within the ductal and vascular systems of the gland before distant hepatic or glandular metastases are evident Accordingly total pancreatectomy is supplemented by excision of adjacent involved tissues Pancreatic gland malignancy has a most unhappy prognosis because of late recognition

Carcinoma of the duodenum resembles other primary gastrointestinal neoplasms in characteristics of growth and spread In definitive therapy therefore the entire vascular arcade and lymphatic drainage of the duo-

denum are excised. Adjacent pancreas and bile ducts need not be widely resected. In discussion of *procedures below, it should not be assumed that* these are restricted to the category under discussion. Anatomical *problems*, clinical status of the patient, individual characteristics of each lesion and other variable conditions will exercise the capacity for adaptability and *technical skill of the surgeon.* Proper preoperative preparation *and post*operative management are also essential to reinforce the extirpation and reconstitution (cf. chapter 9).

Surgical therapy for lesions at the papilla of Vater is loosely *identified as* "pancreatoduodenectomy." This procedure varies so widely in the amount of removed tissues and in reconstruction pattern that evaluation is often equivocal. In addition, benign lesions (which do not usually comprise more than 15 per cent of all tumors in the biliary tract), such as papillomata at the papilla, pancreatitis and carcinoids, may masquerade as malignancy. Results of therapy again are difficult to evaluate.

B. Tumors of Liver, Ducts and Gallbladder

1. Liver

Primary and metastatic tumor in the liver may be resected. The major problem is control of hemorrhage. This can be obtained in several ways:

a. Ligation of the right branch of the portal vein and the right hepatic artery
b. Temporary (15 minutes) compression of vessels in the hepatic pedicle
c. Manual pressure on the liver
d. Mattress sutures through the liver
e. Electrodessication and cautery
f. Gelatin sponge
g. Split thickness skin graft (Masters et al.)

Excision of segments or all of right or left liver lobes has been reported to be successful.

Primary neoplasms of the liver occur in about 0.0175 per cent (autopsy). Metastatic malignancy is found in the liver in up to 40 per cent of all carcinoma up to 35 years after the removal of a primary malignancy.

Tumors of the hepatic ducts may be found in 0.02 per cent of autopsies. These have been found in 30 year old patients. Immediate surgical mortality at present is over 60 per cent. Life expectancy with operation is about seven months and otherwise is about five months. Several patients with duct carcinoma have been reported as well a year after resection.

Surgical excision of liver lobe or segment is hazardous, technically difficult and usually without reward.

Excision of Liver Segment

a Resection of the liver lobe can be done with adequate exposure Although a paramedian incision may suffice for a small segmental resection costal margins and diaphragm should be incised to gain adequate access to the hepatic veins A right thoraco-abdominal extension from a generous transverse abdominal incision is suitable

b Lateral and superior peritoneal reflections of liver to the diaphragm are divided

c On the left

(1) Ligate left hepatic artery

(2) Identify and ligate the left intrahepatic division of the portal vein

(3) Identify and cannulate the left hepatic bile duct

(4) Insert through and through mattress sutures in the interlobar area

(5) Using a knife handle (Quattlebaum) dissect liver tissue away from vessels and ducts these are individually ligated Gelfoam is applied to the exposed liver

(6) Left hepatic duct is intubated and operative area is drained

d On the right

(1) Ligate and cut the cystic duct

(2) Dissect liver hilum to identify and ligate

(a) Right hepatic artery

(b) Right branch of the portal vein

(c) Cystic artery

(d) Cystic duct

(3) Mobilize liver to the left and incise any remaining peritoneal attachments as to kidney and pancreas Gall bladder is removed with the right lobe of the liver

(4) Identify and protect left intrahepatic branch of the portal vein and left hepatic artery

(5) Identify and cannulate common hepatic duct or stump of either right anterior or posterior hepatic duct Ligate if intubation is not possible

(6) Dissect interlobar septum beginning at the falciform ligament preserve a segment of this to cover exposed liver surface Use knife handle (Quattlebaum) to separate liver tissue from vessels and ducts which are individually ligated

(7) Gelfoam omentum skin graft or muscle applied to exposed liver surface

(8) Intubate hepatic bile duct (either intra or extrahepatic) and drain the operative area

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3 Radical Resection of Gallbladder

Excision of right lobe of liver as outlined above is advocated for radical resection of carcinoma of the gallbladder

4 Tumors of Extrahepatic Ducts

✓ Benign papillomata granulomata and other benign and malignant lesions have been removed by local excision from within the common duct In addition since Child and others have demonstrated resection of the portal vein to be feasible, certain lesions of the common bile duct which invade the portal vein may be excised

Mobilization of the duodenum and pylorus are essential to adequate exposure of the common bile duct The posterior aspect of the pancreas should also be mobilized to determine whether pancreatoduodenal resection is indicated

If the portal vein is compressed by the tumor, its ligation may be indicated and one or two stage resection done (Child) As a primary procedure during resection of malignancy, should the lesion be near the distal termination of the duct, pancreatoduodenal resection may be elected In the region of the cystic hepatic common ducts' confluence segmental excision of the duct inversion of the distal end and hepatic duct jejunostomy may be done Cholecystectomy is also done

C Tumors of the Papilla

Differential diagnosis between fibrosis and benign and malignant tumor at the papilla is made more difficult by frequent doubt as to whether a lesion originates in the bile duct the pancreatic duct the papillary tissue or in the duodenal mucosa Frozen section examination may differentiate duodenal biliary and pancreatic origin to malignancy Radical resection is not indicated in the absence of proof for the presence of malignancy

Halstead (1900) reported successful local excision of a carcinoma at the papilla of Vater His approach was transduodenal following the excision he re implanted the ducts into the posterior duodenal wall This procedure is effective (fig 91)

In 1907 Desjardins and Suave suggested excision of duodenum together with the papilla, this was done in 1912 by Kausch, whose patient survived a two-stage procedure only to succumb to a cholangitis after one year

1 Local Excision of Papilla

Mortality for this procedure should not exceed 10 to 12 per cent This procedure is done through combined transcholedochal and transduodenal approaches An opening in the common bile duct is made initially, through which a catheter or probe is threaded towards the papilla (chapter 12) The

duodenum is mobilized Duodenotomy is done to bring the papilla into view (fig 77)

a Lachrymal duct probes are passed through the papilla to identify the duct orifices

b Incision is made into the duodenal wall to circumcise the papilla starting at the mucosa submucosa of the posterior wall through the duodenotomy (fig 80)

c Extraduodenal common bile duct is inspected at its entrance into the duodenal wall hiatus pancreatic gland and capsule are dissected away from it

d The vascular plexus surrounding the duodenal hiatus for the common duct is secured by clips or ligatures

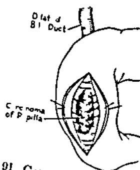


FIG 91 CARCINOMA OF PAPILLA

Drawing of papilla seen at operation which is greatly enlarged by carcinoma Patient died from coronary occlusion 9 days after excision of papilla

e The papilla is dissected from the adjacent duodenal wall to expose the extraduodenal common bile and pancreatic ducts (fig 80)

f Stay sutures are placed in the common bile and pancreatic ducts (fig 80)

g The common bile duct is transected hemostasis is secured

h The pancreatic duct is transected hemostasis is secured

i The lateral aspect of common bile duct is joined to the lateral aspect of the posterior duodenal wall by through and through sutures A long limbed tube or catheter is passed to emerge through supraduodenal choledochostomy

j The medial (left) wall of the pancreatic duct is joined to the full thickness of the left side of the opening in the posterior duodenal wall by through and through sutures

k The duodenal wall between the pancreatic and common bile ducts is approximated in a horizontal pleat by two rows of sutures one being through the entire wall the inner layer approximating the mucosa

l Several horizontal mattress sutures (tied loosely) may be passed to include the ducts and the duodenal wall between them
m Mucosa-to-mucosa sutures are placed to form a septum between pancreatic duct-duodenal mucosa and *common bile duct-duodenal* mucosa (fig 80)
n A catheter is inserted into the pancreatic duct It is retained by a catgut suture
o Duodenotomy opening is closed transversely
p Gastroenterostomy *is established if the duodenum is distorted* A feeding jejunostomy is established if there be any doubt about postoperative alimentation

2 **Radical Excision of Papilla of Vater**

a *Separate the right side of the gastrocolic omentum and the hepatic* colon peritoneal attachment in order to mobilize the hepatic flexure downward
b Continue dissection at inferior aspect of the pyloroduodenal junction and separate gastrocolic omentum throughout its entire extent
c *Identify and free adhesions between the posterior gastric wall and the* anterior pancreatic capsule The mesenteric vessels and vena cava are identified and protected
d The peritoneum to the right of the descending limb of the duodenum is incised The duodenum and the head of the pancreas are reflected *from the posterior abdominal wall The posterior portion of the com*mon bile duct the junction of superior mesenteric vein with the portal vein and the vena cava are identified
e The duodenum and head of pancreas are placed under tension, the cystic and hepatic pedicles are dissected to free the bile duct from adjacent vessels *Dissection then continues superiorly in the liver* hilum
f The head of the pancreas, the duodenum and stomach are retracted downward and a *finger is passed under the posterior* wall of the stomach The lesser omentum is incised and ligated from the left gastric artery to the *pyloroduodenal junction The right gastric artery*, the hepatic artery, the gastroduodenal artery and the anterior and posterior branches of the pancreaticoduodenal vessels are exposed (A thorough evaluation is done at this time before continuing There has been no committment and the procedure may be now interrupted)
g *The plane between the pancreas and portal vein should be freed gently* The portal vein may be ligated
h Branches of the vascular arcade involving the superior pancreatoduodenal and the inferior pancreatoduodenal, the right gastric, *the left*

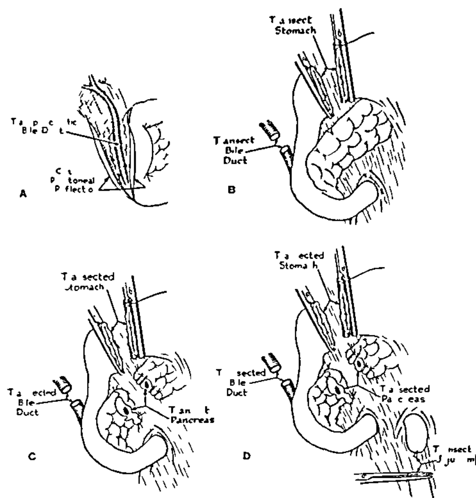


Fig 92 Diagrams of Principal Steps in Pancreatoduodenal Resection

A Identify retroduodenal common duct portal vein arterial arcades and pancreas after incision of lateral duodenal reflection and mobilization (to the left) of the descending duodenum

B Transect stomach proximal to pyloric antrum Bile duct is cut at site which will avoid compromising the gallbladder and its circulation (In case of low implantation by the cystic duct the septum may be excised Cholecystectomy may be required)

C Transect pancreas Suture ligatures are placed to obtain hemastasis

D The duodenum is transected distal to ligament of Treitz

gastric and the right gastro-epiploic vessels are secured The mesocolon is elevated The jejunum is identified and cut to provide for its greatest mesenteric length (fig 92)

1 The proximal jejunum is closed by Parker Kerr suture and withdrawn

through the mesocolon opening to be removed en masse with the resected tissues. The distal cut end of the jejunum is temporarily replaced after covering with a sponge beneath the mesocolon. (This will either be brought through the mesocolon or over the right side of the hepatic flexure in restoring gastrointestinal continuity.) Dissection proceeds from left to right and from below upward following the portal vein and vena cava and freeing the pancreas from its adjacent vessels.

j. The margins of the pancreas are dissected back to the first part of the body and in doing so the inferior pancreatoduodenal artery and veins are ligated. Anastomotic vessels between the upper margins of the body of the pancreas and the splenic vessels are ligated. Section the pancreas and place a rubber catheter in the pancreatic duct. Mattress sutures or figure of eight sutures are placed in the body of the pancreas to secure hemostasis and to prohibit enzyme loss (fig. 92).

k. The pyloroduodenum is freed. The mass is mobilized upward and lifted across to the left side of the incisional area. Hemostasis is obtained.

l. The common bile duct is cut at the highest useful point. The cystic duct and artery are ligated and cut, the gallbladder is removed.

m. A hole is now made in the mesocolon in an avascular region and the jejunum (at least 18 inches) brought through it. The open jejunum is anastomosed to the hepatic end of the common duct.

n. Remaining pancreas is attached to the jejunum by end-to-side anastomosis.

o. The long loop of jejunum is then brought over to the lesser curvature of the stomach where it is angulated and a partial obstruction to its lumen produced.

p. The entire length of the posterior gastric wall is then sutured to the jejunum.

q. Stomach is cut and aspirated. The malignancy is removed. The anastomosis of the stomach to the jejunum is then completed.

r. Redundant jejunum is attached by side-to-side anastomosis below the gastrojejunostomy in order to permit the bile and pancreatic juices to drain directly into the jejunum.

s. A Babcock sump drain is placed.

t. A feeding jejunostomy may be utilized.

Variations starting at "m" above:

(1) A Roux en Y anastomosis may be accomplished by end-to-end anastomosis of the common duct to the jejunum.

(2) Jejunum may now be cut and end-to-end anastomosis done between the jejunum and stomach.

(3) End to side jejunostomy is then done to complete the "Y." A tube may be placed through the common duct into the jejunum to emerge through a separate incision in the common duct away from the anastomosis
(4) The choledocho jejunal anastomosis is anchored to the liver hilum
(5) The lesser curvature of the stomach may be closed and the specimen removed prior to the gastroenterostomy which is done along the greater curvature side

D Circumductal Lesions

Benign lesions of the papilla and pancreas may be of sufficient extent to warrant limited pancreatoduodenectomy Certain doubtful lesions in the head of the pancreas those of proven malignancy in the head of the gland, in the patient who has too prolonged anesthesia time or whose general condition is deteriorating these require staged procedures or a limited resection In such cases less than half the pancreas, stomach, extraduodenal common bile duct and first three portions of the duodenum may be removed

1 Resection of the Head and Body of the Pancreas

a The lateral duodenal reflections are incised, the duodenum and head of the pancreas are mobilized to the left (fig 71)
b The gastrocolic omentum is ligated and cut to just below the vasa brevia
c The hepatic and transverse colon are mobilized downward
d The stomach is elevated Inferior aspect of the pancreas is mobilized identifying the superior mesenteric vessels and the portal vein
e The middle colic artery is identified and protected
f The gastrohepatic omentum is ligated and the fundus of stomach placed on tension Dissection continued in the gastrohepatic omentum towards the liver The right gastric artery and gastroduodenal artery are ligated
g The portal vein and common bile duct are exposed down to the head of the pancreas
h The stomach is transsected The lesser curvature is closed by an inner row of chromic catgut and an outer row of interrupted silk sutures This is reinforced by the gastrohepatic omentum
i The common bile duct is cut
j The gallbladder is aspirated and a Pezzer catheter inserted with purse string sutures for cholecystostomy
k The pancreas is transsected Posterior border of the pancreas is freed from the portal vein

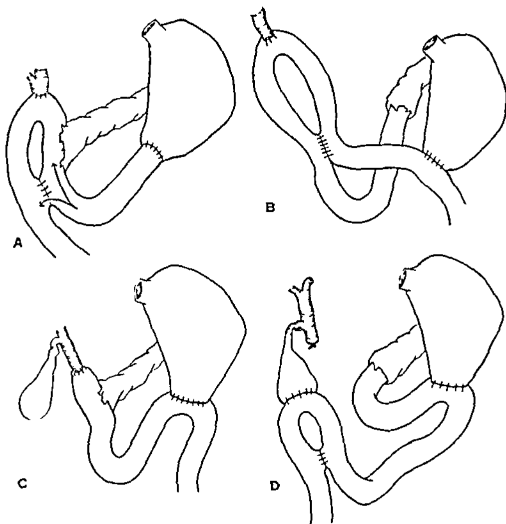


FIG 93 METHODS FOR RESTORING CONTINUITY AFTER PANCREATODUODENAL RESECTION

Arrows indicate direction of flow

A Gastrojejunostomy enteroenterostomy choledochojejunostomy and pancreatojejunostomy in the jejunal loop

B Pancreatoduodenostomy choledochojejunostomy enteroentero tomy gastrojejunostomy

C Choledochojejuno tomy, pancreatojejunostomy gastrojejunostomy

D Pancreatoduodenostomy gastrojejunostomy enteroenterostomy cholecystojejunostomy in the intestinal loop

l The fourth portion of the duodenum is cut and the specimen removed

m The distal half of the pancreas is approximated to the fourth portion of the duodenum by end to-end anastomosis The posterior duodenal wall is sewn to the posterior surface of the pancreas the anterior duodenal wall to the anterior surface of the pancreas

n A long loop of jejunum is brought in front of the colon On the afferent portion a choledocho enterostomy is accomplished using an inner row of interrupted catgut and outer row of silk sutures

o The jejunum at the site of the choledochojejunostomy is brought up over the common duct to reinforce the anastomosis Reinforcing sutures attach the jejunum to the hepatic pedicle

p A gastrojejunostomy is then done in the efferent loop An indwelling Abbott Rawson tube is brought through the anastomosis to the distal jejunum Gastro enterostomy is completed by an inner row of dis continuous chromic catgut and an outer row of interrupted silk su tures The anastomosis is protected by segments of gastrocolic omen tum (fig 93)

q An entero-enterostomy is done between efferent and afferent loops between the choledochojejunostomy and the gastrojejunostomy using two rows of sutures

r Penrose tube drain is placed near the choledochojejunostomy

2 Circumductal Lesion Originating in the Duodenal Wall

Polypoid or ulcerating carcinoma of the duodenum is not common Rad ical excision includes the entire duodenal vascular arcade Limited segments of stomach pancreas and bile ducts are removed (fig 36)

Operative procedure consists of cholecystectomy, partial gastrectomy, total duodenectomy partial pancreatectomy, partial choledochectomy and re establishment of gastro and biliary intestinal continuity by an end to-side choledochojejunostomy and end to-side pancreaticojejunostomy (over a catheter or a 'T tube) and an end to side gastrojejunostomy, in that order

E Pancreatoduodenectomy

1 Resection

Removal of the termination of the common bile duct other than by local excision and re implantation of the ducts can be done by 81 possible varia tions The stomach duodenum pancreas and bile ducts may each be re moved in three proportions (3^4—81) The possible variations are

a Stomach resection
(1) Prepyloric
(2) Distal third
(3) Distal half or more

b Duodenum resection
(1) First second and third portions
(2) Entire duodenum
(3) Duodenum plus proximal jejunum

c Pancreas resection
  (1) Head (and neck),
  (2) Head, neck and right half of body,
  (3) Total
d Bile ducts resection
  (1) Papilla and distal common bile duct,
  (2) Gallbladder, common bile duct, and papilla,
  (3) Common hepatic and common bile ducts, gallbladder and papilla

Cattell and Warren describe many of these (fig 93), Orr gives their historical background and students of surgery are constantly devising other ramifications It is hoped, in evaluation of results, that more specific description than "pancreatoduodenectomy" will be given in the future It would be informative to indicate, with reference to the possibilities above, that in a patient with tumor of the distal common bile duct a(1)—b(1)—c(3)—d(2) pancreatoduodenectomy was done

Pancreatoduodenectomy for malignancy has an immediate mortality of less than 18 per cent Cattell and Warren report over 100 such cases with 12 7 per cent mortality

Survival time, however, does not indicate an optimistic prognosis In 38 cases of carcinoma of the head of the pancreas (Lahey) one survived six years and one for five years

In carcinoma of the papilla, however, at least 20 per cent of patients may survive for more than five years Carcinoma of the distal common bile duct averages a one year survival Carcinoma of the duodenum averages 18 months In individual cases, survival of six or seven years without recurrence have been reported

Surgical mortality for removal of carcinoma in the peripapillary region averages 12 to 15 per cent However, after five years it is not expected that more than 5 per cent of patients would be alive

These statistics have been improving through the years since Whipple Parsons and Mullens first introduced a method for pancreatoduodenal resection in 1935 Originally, this was staged The preliminary procedure was a cholecystogastrostomy and a gastroenterostomy This was followed by resection of the head of the pancreas and the duodenum with inversion of all cut ends Another type of procedure which Whipple used was a Roux en Y procedure using an end to-end anastomosis between gallbladder and jejunum

Trimble Parsons and Sherman advocated utilization of the cut end of the stomach in anastomosis Hunt first adapted common duct anastomosis in lieu of the gallbladder because the common duct ligature usually did not hold

Hunt and Cattell introduced direct anastomosis of the pancreas and

jejunum Dennis stressed the value of a long intestinal loop between the biliary and gastroenterostomies in order to prevent reflux of intestinal content into the liver Dennis and Varco also advocated implantation of the common duct to the long jejunal limb Recently it has been suggested that high gastric resection be done to avoid late postoperative sequellae However, justification for both entero anastomosis and high gastric resection have not yet been fully established

2 Reconstruction

There are several factors concerning reconstructive procedures which affect optimal function First it is desirable to prohibit flow of chyme and gastric content into the bile and pancreatic ducts It is definitely advantageous to have peristalsis carry all material caudad

Second the common bile duct (or hepatic duct) is preferred to the gallbladder for anastomosis following pancreatoduodenectomy The proximal end of the common bile duct should be used in order to avoid fistula formation In addition there may be low implantation of the cystic duct in the common bile duct A septum may be present at the site of anastomosis There is no objection to leaving in the gallbladder and doing an additional anastomosis between the gallbladder and the jejunal loop or establishing a cholecystostomy

Third if any pancreas remains it should be anastomosed with available small bowel The usefulness of the pancreatic ferments is undoubted In addition pseudocyst abscess or fistula may appear unless the pancreato-intestinal continuity is restored

There are at least 36 suitable methods for reconstitution after one of the types of gastro-duodeno-pancreato-choledochectomy (3X3X2X2)

a Stomach to jejunum [1]
   (1) end (part or all) to proximal end
   (2) end (part or all) to end (of Roux en Y)
   (3) end (part or all) to side (of loop)
b Pancreas to jejunum
   (1) duct and gland to wall (necrotizing)
   (2) duct and gland to mucosa and wall (tube)
   (3) gland to bowel (open)
c Biliary tract to jejunum
   (1) common duct end to-end
   (2) common duct end to-side
d In all cases a complementary entero-enterostomy may be done

[1] Side to side and side to end anastomoses introduce additional suture lines and are not satisfactory

[2] Useful as adjunct but not dependable as primary anastomosis is gallbladder anastomosis to end or side of jejunum

Reports of reconstruction could well be identified in the *order of flow* and in the character of the anastomosis For example, 'c(1)—b(2)—a(3)' could be established following resection for tumor of the *common bile* duct In this case an additional note could identify the presence of an entero-anastomosis in the route as follows "c(1) LE b(2) a(3)'

Some method for simple identification is needed in order to evaluate various methods for reconstruction

F Palliative Procedures

Radical resection for lesions of the common bile duct including the papilla is not always *indicated nor feasible In many cases when there is* doubt concerning presence of malignancy it is preferred to by pass the *obstruction In others just as it appears that a radical excision could* have been done, either an excessive fixation to the mesenteric vessels is *observed or the patient's condition under anesthesia deteriorates and a* palliative procedure is elected Life can be prolonged, digestive and other *symptoms relieved by palliative procedures*

There are three obstructing mechanisms to be relieved First, the biliary *tract second, the pancreatic duct and, third, the intestinal tract Accord*ingly adequate palliation for an unresectable lesion at the papilla should *include gastrojejunostomy, cholecysto (or choledocho-) jejunostomy* and pancreatic duct jejunostomy

1 Decompression

Certain drainage procedures are often necessary since the patient's condition may not permit even a "by pass' procedure The y include

a *Hepaticostomy (intrahepatic duct drainage)*

b Choledochostomy (includes also all extrahepatic ducts)

c *Pancreatic duct drainage*

The insertion of a catheter or ' T" tube is advantageous *Intubation is an excellent guide in returning to the area*

2 Pancreatic Duct-Jejunostomy

a Side to-side

b *End of-duct to side of jejunum*

c End of-duct to end of jejunum

d *Side of-duct to end of jejunum*

The pancreatic duct may be dilated by obstruction at the head of the *pancreas or in the papilla It can often be palpated* The duct is exposed by incision into the gland after separation of the gastrocolic omentum

Ordinarily a side to-side pancreatic duct jejunal anastomosis can be constructed This should be supplemented by an entero-enterostomy be

tween the afferent and efferent limbs In some cases the duct cannot be identified or has been damaged In such cases the gland is cut and the body anastomosed to a defunctionalized limb or loop of jejunum The head end of the gland may be oversewn

3 Gastro enterostomy

Duodenal obstruction is very common in the late stages of peripapillary malignancy Additional manipulations in the descending limb of the duodenum at operation may precipitate pyloroduodenal obstruction Accordingly if any by pass procedure is attempted such as will relieve hepatic obstruction supplementary relief of impending or actual pyloroduodenal obstruction should also be done An antecolic or retrocolic loop of jejunum is anastomosed to the greater curvature Should pancreatic duct jejunal anastomosis also be done the latter is placed in the distal efferent loop of the gastrojejunostomy

4 Biliary Duct By pass (Fig 90)

a Cholecystogastrostomy
b Cholecystoduodenostomy
c Cholecystojejunostomy
d Choledochal anastomoses

In the presence of a distended gallbladder when diagnosis is not certain or when the primary lesion has widely metastasized anastomosis between the gallbladder and the intestinal tract may be done This is a suitable procedure provided that the cystic duct is patent and its implantation is high and that the gallbladder fundus and intestinal loop can each be mobilized to avoid tension

a Cholecystogastrostomy
(1) The gallbladder peritoneum is incised to mobilize it from the hepatic bed
(2) The gallbladder is aspirated through an incision on the fundus which is selected for use as an anastomotic stoma Occasionally the anterior wall or the lesser curvature of the stomach will be anatomically adjacent to the distended gallbladder
(3) Procedure is as in the usual side to side intestinal anastomosis However the opening in the gallbladder should be larger than that in the stomach In addition the anastomosis should have two rows of interrupted non absorbable sutures

b Cholecystoduodenotomy
(1) When a duodenotomy has been done for extirpation of the papilla this duodenotomy opening may be utilized for anastomosis to the gallbladder

(2) The cholecystic-duodenal anastomosis is accomplished by two rows of interrupted non absorbable sutures. The gallbladder, as it decompresses, tends to shrink away from the duodenum and also tends to close the anastomotic stoma. It may also partially occlude the duodenal lumen. (Use of the interrupted suture tends to maintain the patency of the stoma.)

(3) The cholecystoduodenal anastomosis is made more secure by attaching the lateral duodenal peritoneal reflection to the gallbladder peritoneum. In addition, a complementary gastroenterostomy may be done.

c. Cholecystojejunostomy. Use of cholecystojejunostomy is feasible if the stomach is anatomically unsuitable, if the mesocolon and omentum are not too fatty and if the jejunum is conveniently available. Either a defunctionalized loop or a limb may be used. Interrupted non absorbable sutures are used. If the loop of jejunum is used, a complementary enteroanastomosis is usually done. This prevents kinking of the jejunal loop by shrinkage and displacement when the distended gallbladder has been decompressed.

d. Choledochogastrostomy, choledochoduodenostomy and choledochojejunostomy (loop or limb).

When junction of cystic and common ducts cannot be identified or if it is seen that the cystic duct is implanted low on the extraduodenal bile duct or if the obstructing lesion is close to the cystic duct, an anastomosis between the gallbladder and intestinal tract will function inadequately and only for a brief period.

Accordingly, a suitable part of the common bile or hepatic duct is selected for anastomosis. In some cases the duodenal loop is immediately adjacent or has been mobilized by incision of the lateral peritoneal attachments.

The usual side-to-side anastomosis is done. However, duct anastomosis to stomach or duodenum is not as useful in malignancy as it is in patients with stricture or fistula. Accordingly, a *defunctionalized loop or limb* of jejunum may be brought to the hepatic duct for anastomosis. The limb is preferred because of the extra length it gives to reach the liver hilum and the resultant *freedom from tension*.

Duct to bowel anastomosis may be constructed over an indwelling tube.

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ing This may be due to loss of blood volume into the liver into dissected tissue planes or into the mesenteries Shock may be due to pancreatitis or bile peritonitis

2 Bile Peritonitis (Chapter 4)

Among causes for postoperative shock are a slipped ligature from the cystic duct or leakage from an unobserved accessory duct, particularly in the gallbladder bed The effect of spillage of bile during operation is negligible However uncontrolled continued leakage of bile intraperitoneally may be fatal Bile leakage should be controlled by ligature packing and drainage if it does not subside within 24 hours

3 Duct Obstruction

Differential diagnosis of obstructive jaundice following biliary tract surgery rests among (a) organic disease or calculi in the common duct (b) trauma to the common duct including partial or circumferential ligature, (c) hemolysis (d) cholangiolitis or hepatitis and (e) edema, mucus or hyperemia which induces transient obstruction at the papilla or in the common bile duct

Reoperation to relieve continuing increasing icterus is not urgent It may take ten days to establish a positive diagnosis or to determine that a benign or inflammatory process alone is present

Relief of duct obstruction is required to decompress the liver Maintenance of a route for bile into the intestinal tract is not as important as the relief of obstruction Free flow of bile is manifest by subsidence of bilirubinemia and by the appearance of bile in the feces of urobilin in the stool and urine and the disappearance of bile from the urine All these should occur with relief of obstruction If not, the patient may have a type of icholia associated with hepatic failure to produce bile (chapters 4 and 6)

4 Ileus and Intestinal Obstruction

Inexpert manipulation technical inefficiency coincident appendectomy, preexisting adhesions leakage of bile or other enzymes reflex neurogenic stimuli and postanesthesia effects may inaugurate an ileus or intestinal obstruction

Gastric distention occurs due to the pyloroduodenal manipulations This is manifest by repeated vomiting of brown fluid associated with left abdominal and subcostal pressure and distention Gastric dilatation may be overlooked It is an important cause of later evisceration as well as of electrolyte imbalance Correction is accomplished by a nasogastric tube, using continuous or intermittent suction

Failure to correct the distention by use of the nasogastric tube and continuing evidence of bowel obstruction may require decompression by a long intestinal tube (Cantor, Harris or Miller Abbott) Operation to relieve obstruction to the bowel is occasionally indicated

Following cholecystojejunostomy reoperation may be required to establish a complementary enteroenterostomy Pyloric obstruction may require gastroenterostomy Other indications for reoperation in the presence of intestinal obstruction are individual problems

B Liver Biopsy

Examination of liver tissue is very valuable to supplement other laboratory methods for evaluation of hepatic function Microscopic recognition of cirrhosis malignancy and hepatitis may solve the problem of jaundice It is essential that prothrombin bleeding and clotting times be normal and that blood be available for transfusion

The liver mass is outlined by physical examination or by radiography The patient is given adequate sedation Biopsy may be obtained directly by Silverman needle (fig 94) aspiration or through the peritoneoscopy

The patient remains in bed for 8 to 24 hours after the procedure The longer interval is used if localized tenderness or rigidity are associated with rebound phenomena These indicate peritoneal irritation possibly due to leakage of blood or bile No serious sequelae such as peritonitis thrombosis atelectasis, pneumonitis or pneumothorax have been observed following liver biopsy Bleeding has been the only major problem

C Infection

1 General

Identification of etiologic bacteria is essential in therapy of infection The use of an antibiotic is selected on the basis of specific sensitivities Caution is necessary however in placing complete reliance on a single laboratory report Occasionally an *in vivo* sensitivity may not be demonstrated in the Petri dish

General management of patients who have infection following bile duct surgery is as follows

a Sulfonamides

(1) bacillary infections of urinary tract—1 gm every 4 hours (Gantrisin)

(2) chronic bacillary bronchopulmonary infections—1 gm every 3 hours (Gantrisin)

(3) intestinal tract infection (or prior to second stage of pancreatoduodenectomy) Sulfasuccidine Sulfathaladine 2 gm 4 times daily

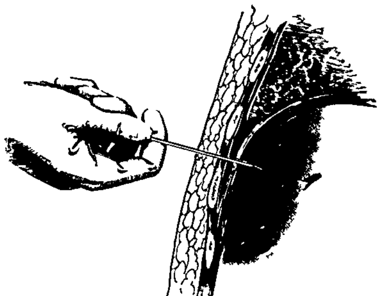


FIG. 94 A

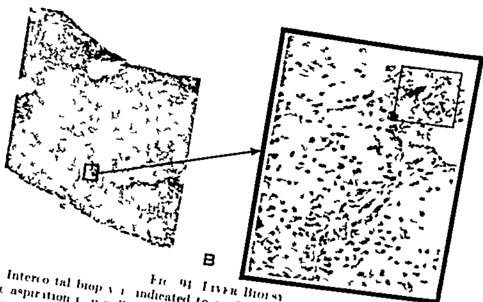


FIG. 94. LIVER BIOPSY

A. Intercostal biopsy is indicated to confirm or establish diagnosis. Silverman needle aspiration is usually uneventful. Special precautions are taken in patients with suspected abscess and in those with hypoprothrombinemia. (Courtesy of Charles Pfizer & Co., Inc.)

B. Liver biopsy specimen (low power) reveals histologic evidence of (chlorpromazine in this case) cholangiolitis. Under high power in the small square bile pigment lakes have extravasated around cholangiole. In this section there is minimal hepatic cellular change and no necrosis (cf. Figs. [illegible])

b Penicillin—600 000 units procaine penicillin G intramuscularly daily
c Aureomycin Terramycin and Achromycin —may be used interchangeably—0 25 to 0 5 gm every 6 to 8 hours
d Chloramphenicol—0 5 gm every 3 hours until temperature is normal then every 6 hours orally
e Polymyxin B—50 to 100 mg intramuscularly 3 times daily
f Erythromycin—0 25 to 0 5 gm every 4 hours (oral or intramuscular)
g Bacitracin—25 000 units every 6 hours first day then 3 times daily intramuscularly
h Streptomycin and Dihydrostreptomycin—0 5 to 1 5 gm every 24 hours

2 Subdiaphragmatic Abscess

Conservative management with antibiotics is valid except where constitutional and localizing symptoms indicate the present of (undrained) pus under pressure In an empyema of the gallbladder or severe cholangitis an effective concentration of the antibiotic cannot usually gain access to the involved area because of calculous obstruction (Zaslow) Under such conditions operative drainage is the least procedure which is required

Postoperative abscess may occur in an inadequately drained subdiaphragmatic space

Surgical approach to the right anterior space may be through a muscle splitting incision below and parallel to the costal margin Incision is carried to the peritoneum which is mobilized upward to reach the abscess Drainage is established through muscle planes to avoid gross contamination of peritoneal and thoracic cavities

Ochsner and others have described an approach to the right posterior superior and inferior spaces through the bed of the resected twelfth rib Blunt dissection separates pleural and peritoneal membranes until the subphrenic space is entered The abscess is usually adherent to the parietal peritoneum For the other subdiaphragmatic spaces anterior extraperitoneal approaches are suitable

When biliary tract abscess has ruptured through the diaphragm and has established a biliary pleural or biliary bronchopleural fistula drainage alone may be insufficient In such instances persistence of the abscess may be due to co-existent biliary tract disability such as from a cholangitis In this type of case choledochostomy with provision made for adequate bile flow through the normal ductal system is essential to permanent relief (Hewlett)

D Portal Vein Complications

Since the portal vein is adjacent and posteromedial to the common bile duct it may be injured during operative procedures involving the biliary

tract In secondary operations on the bile duct extensive adhesions may obliterate the bile passages It is not unknown therefore that the portal vein may be incised and its intubation be undertaken mistakenly

Such incision need not be catastrophic The portal vein can be repaired using interrupted everting sutures of fine silk The Pott's and Blalock instruments are very useful

If the portal vein has been invaded or partially occluded by malignancy it is not necessary to abandon the resection (Child) When the portal vein pressure is high it is recommended that the portal vein be ligated and cholecystostomy or choledochostomy be done and that second stage resection of the portal vein (and the neoplasm) be accomplished after eight to ten days If feasible at the first stage, splenectomy or portocaval shunt may be done Splenectomy is indicated because ligation of the portal vein frequently inaugurates splenic vein thrombosis and in most cases collateral venous drainage of the spleen is inadequate

If portal vein pressure is low, resection of the portal vein may accompany the primary operation The mesenteric and splenic veins may be ligated on the inferior and medial surfaces of the pancreas Splenectomy may also be required

Child measures portal vein pressure before and after ligation of the vein and does a concomitant portal venogram to indicate the presence of an adequate collateral circulation If the portal venogram has a subtraction defect and the portal pressure is normal the portal vein can be obstructed by a Blalock clamp The portal vein pressure is again measured after 20 or 30 minutes If there be only a slight rise in portal pressure it can be assumed that sufficient collateral exists However if after occlusion the pressure rises to 35 to 40 cm of saline it is best to perform the operation in two stages The major reasons for deferring to a two stage procedure are first that an inadequate collateral circulation may produce mesenteric thrombosis and gangrene and second that it is extremely hazardous to operate through a sanguineous field originating in and augmented by increased venous pressure

Splenectomy is indicated if the vessels are damaged and if total pancreatectomy is done However since useful anastomotic channels are provided by splenic routes to the omentum stomach and other viscera careful consideration is to be given to permitting the spleen to remain In addition should portal hypertension remain a problem after pancreatoduodenal resection with ligation of the portal vein the splenic vein can be utilized later as a splenorenal shunt

Damage to the bile duct during portocaval shunt has not been reported In most cases it is not difficult to mobilize the common bile and hepatic ducts in order to gain adequate exposure and mobilization of the portal

vein In several in tances however the bile duct has been lacerated or inadvertently incised In such circumstances a T tube or drain should be inserted to control bile seepage which might deleteriously affect the vascular anastomosis

E Electrocoagulation Technique

Electrocautery and electrodessication techniques are suitable for subtotal cholecystectomy when it is impossible to identify the relation between the common bile and cystic ducts Under such circumstances the cystic duct and artery are ligated by mass suture the gallbladder mucosa is obliterated by cautery or the outer wall has its mucosa stripped and is then coagulated or dessicated (Pribram Thorek Estes) This area can be covered by omentum or falciform ligament Estes has improved this procedure of partial cholecystectomy which is accomplished by surgical dissection of the inner lining away from the gallbladder wall and its approximation and obliteration by suture In all these ligation of the duct and artery are attempted

Electrodessication cholecystectomy done when visibility of the triangle of Calot is zero is best accomplished when a catheter is passed through the cystic duct orifice to remain and drain postoperatively The catheter primarily provides additional assistance in dissection secondarily drainage of the common bile duct thirdly postoperative cholangiography can be done and fourthly, it acts as a geographical marker if reoperation is required

When the operative field is not too close to the vena cava or the portal vein there is no objection to the use of electrosurgery in dissection

Excision of a liver segment is abetted by mattress sutures reinforced by electrocoagulation and dessication This technique should supplement individual ligation of large ducts and vessels Secondary bleeding may occur for ten days after electrocoagulation

In areas wherein ligation or suture ligation are not feasible the electrocoagulating current may be helpful The silver clips used in neurosurgical procedures also are advantageous for hemostasis

F Drainage of the Pancreas and Its Ducts

Coincident disease of the pancreas is often found with biliary tract disorders Correction of abnormalities in the bile ducts may simultaneously effect relief to dysfunction in the pancreas For example those cases of chronic choledocholithiasis associated with an (abnormal) interductal fistula may be relieved by prolonged choledochostomy

In many cases drainage is required to relieve pancreatic suppuration and cystic collections and to ameliorate progressive disabling symptoms

In definitive therapy to certain pancreatic cysts adequate drainage of the pancreatic duct through the papilla is essential (Doubilet)

The patient with pancreatic calculi or calcinosis does have disability from pain In most cases the discomfort is not enough to substantiate the risk of total pancreatectomy Drainage of the duct and the pancreas is more suitable This may fully relieve symptoms

1 **Pancreatolithotomy**

a Transpapillary Intubation of the Normal Pancreatic Duct is Difficult

The escape of a small amount of pancreatic juice will identify the orifice If the pancreatic duct orifice is identified, then incision through the medial aspect of the papilla will overcome sphincter activity surrounding the pancreatic duct The duct is then intubated with a rubber catheter or polyethylene tube The catheter may be passed through a duodenotomy if a side opening is made to permit pancreatic juice to enter the duodenum Otherwise the tube is sewn into place with fine silk and drains into the duodenum After the knot has digested normal intestinal peristalsis will remove the tube from the pancreatic duct to be passed with the fecal current

It is possible to combine transpapillary drainage of the pancreatic duct with drainage of the pancreas (duct gland or cyst)

Pancreatic calculi may be removed by transpapillary approach The pancreatic duct should then be injected with radio-opaque medium for radiologic visualization Residual calculi should be removed or provision made for improved flow to the pancreatic enzymes

Transpapillary exposure of the pancreatic duct is al o done at the time of excision of the papilla (chapters 12 and 13) In doing this an internal strut, such as an ureteral catheter is valuable and frequently essential Midway between sphincterotomy and resection of the papilla of Vater is the procedure of wedge or segmental excision of the papilla This is accomplished by removing a portion of the medial aspect of the papilla in order to establish unhindered flow from the pancreas

b Pancreatolithotomy or Drainage of the Pancreatic Duct Is More Commonly Attempted in the Body or Tail of the Gland

In dealing with benign disease of the pancreatic ducts and glands several alternative procedures are available 1) Simple removal of calculi and drainage of the duct by intubation 2) Anastomosis of the duct to a defunctionalized loop or limb of small intestine 3) Resection of the proximal or distal pancreas with anastomosis of remaining segments to defunctionalized loop or limb of bowel 4) Any of the preceding plus establish

ment of adequate flow through the papillary portion of the pancreatic duct by means of approach through duodenotomy

Anastomosis of the dilated pancreatic duct to the jejunum is also indicated as palliative therapy to relieve obstruction due to unresectable malignancy at the papilla Approach to the pancreatic duct in the body of the pancreas is usually accomplished during the exploratory and mobilizing phases of attempted pancreatoduodenectomy Should the resection be abandoned it is not difficult to apply an available jejunal loop to the pancreatic duct Otherwise incision of the gastrocolic omentum with separation of the stomach and colon and with particular care regarding protection to the middle colic vessels will expose the body of the pancreas

c Pancreatic Duct Drainage in Some Cases Can Be Effected Near the Splenic Pedicle by Mobilization of the Gastrocolic Omentum on the Left

The pancreatic duct is very small at the tail of the gland It is however feasible to establish tube drainage or intestinal anastomosis between duct and bowel using the transected tail of the pancreas

Caudal pancreatostomy is a feasible palliative procedure when the pancreatic duct is greatly dilated because of calculi It is made more effective by transpapillary drainage of the pancreatic duct Only rarely are both caudal and transpapillary methods of duct pancreatostomy indicated simultaneously

2 Pancreatostomy

Suppuration and necrosis in a pancreatic abscess may reach huge proportions Pancreatic enzymes can convert an abscess to a pseudocyst Drainage of a large collection can relieve symptoms and may halt progress of the disease In the majority of cases however the drainage tract becomes a permanent fistula Useful drainage for the head of the gland is obtained by right lateral approach Anterior transabdominal drainage is used for the body and tail of the gland Left retroperitoneal drainage (12th rib bed) similar to drainage for subdiaphragmatic abscess is utilized for posterior collections Because of the frequency of fistulation drainage or marsupialization procedures are considered a preliminary to later definitive therapy

a Right Lateral Approach to the Head of the Gland Is Much the Same as Approach for Mobilization of the Duodenal Loop (Kocher)

In such cases the foramen of Winslow is usually occluded by adhesions and exudate Mobilization of the duodenum may be difficult and hazardous Necrotic suppurative and calcified masses may be scooped from an abscess behind the duodenum and stomach Drainage is maintained by a sump

drain Secondary therapy to papilla bile ducts or other areas depends upon associated disease

b Anterior Drainage for Pancreatic Collections Always Appears to Be Easy Since the Fluid Collection Gives the Impression of Being Subcutaneous

However, any needle aspiration is contraindicated Either the stomach or the colon is nearly always between the pancreatic abscess or cyst and the abdominal wall Anterior drainage is accomplished by mobilizing the stomach (upward or downward) depending upon its position relative to the collection Incision in the gastrocolic or gastrohepatic omentum is necessary

c Posterior Approach to a Pancreatic Abscess Is through the Bed of the Left 12th Rib Just as If It Were Subdiaphragmatic Abscess Which Indeed It Is

Drainage by this route is desirable since peritoneal contamination usually is not present and through this approach is difficult to produce

3 Pancreatic Cysts

Surgical treatment is preferentially indicated in pancreatic cysts Conservative management usually results in death from cachexia or spontaneous rupture of the cyst Excision is the ideal method of treatment Unfortunately it is feasible only in approximately 25 per cent of the cases

By draining the cyst into the digestive tract, fluids electrolytes and enzymes contained in the cystic cavity are reabsorbed and the skin excoriation of external drainage is obviated Pancreatic cysts can be anastomosed to stomach, jejunum or gallbladder

Approach to the cyst may be transgastric (Jurasz) When a pseudocyst is intimately bound by adhesions to the posterior gastric wall, transgastric drainage can be established through a simple opening of the area adherent to the stomach without performing a two layer anastomosis A stoma at least 4 to 5 cm in length is needed to insure adequate drainage, especially in cases of pseudocysts where the lack of an epithelial lining favors premature obliteration of the stoma

Anastomosis of a jejunal loop to the cyst can be achieved either according to the principles of the usual gastroenterostomy or by adding a jejunojejunal anastomosis to the cystojejunostomy When a defunctionalized limb is used this is usually brought through the transverse mesocolon

The pseudocyst which follows pancreatic necrosis or inflammation or which is associated with biliary tract disease is not to be confused with the malignant cyst or the traumatic developmental or parasitic types

Developmental and traumatic cysts may respond to the same procedures

used in treatment of the pseudocyst Malignant and parasitic cysts however are best excised or adequately marsupialized without intraperitoneal or intestinal contact

G Cysts of the Biliary Tract

Choledochus cysts of congenital origin are rare The treatment of the cyst in the absence of jaundice is anastomosis to the adjacent duodenum or to a physiologically isolated loop or limb of jejunum (Shallow) Appropriate therapy of the obstructive jaundice is indicated if the anastomosis fails to relieve this Cholangiography is valuable

H Annular Pancreas and Pancreatic Heterotopia

Annular pancreas is a benign condition Resection maneuvers are too great a risk for its relief In such cases duodenoduodenostomy gastroduodenostomy or gastrojejunostomy are indicated Should jaundice be present an appropriate biliary by pass may be established

Pancreatic heterotopia occurs in the stomach bowel gallbladder bile ducts duodenum and spleen in addition to other very rare sites Its recognition depends upon suspicion and upon frozen section examination It is most often mistaken for malignancy When recognized no therapy is required Areas which may become inflamed in strategic locations such as in the common or cystic ducts may produce alarming symptoms

I Duodenal Diverticula

Since two thirds of duodenal diverticula occur in the duodenal hiatus for the papilla of Vater is not unexpected that the papilla on occasion may empty directly into a diverticulum In addition the presence of a peri papillary diverticulum may complicate duodenotomy and sphincterotomy

Since there is usually no pathognomonic symptom complex a duodenal diverticulum may well be an unexpected and confusing cause for the post cholecystectomy syndrome It may obstruct the bile or pancreatic ducts or the duodenum There may be inflammatory neoplastic or hemorrhagic changes within the diverticulum

The fecal current is liquid in the duodenum Therefore Ferguson (1947) recommended inversion of the diverticulum as an extremely satisfactory maneuver In other cases the diverticulum is excised and the defect is closed in layers Closure is often very difficult after excision of the diverticulum because of the many blood vessels entering the duodenal wall at the location of the sac s neck

Although radical pancreatoduodenectomy has been reported being done for diverticulosis of the duodenum such procedure usually was done because of a mistaken diagnosis of malignancy

Surgical repair of the peripapillary diverticulum requires mobilization of the duodenal loop and intubation of the bile duct (MacLean)

J Duodenal Stump

Biliary tract surgery may be required subsequent to gastrectomy The duodenal stump, after mobilization, may be utilized for anastomosis with the hepatic duct The anastomosis should be anchored with non absorbable suture to or through the liver in order to avoid tension

K *Denervation Procedures*

Certain individuals, particularly those who have symptoms following *cholecystectomy, may have spastic or atonic dyskinesia Different char*acteristics are due to physiologic mechanism within the bile ducts and are activated by many trigger points" (chapter 15)

Analysis of results after right splanchnicectomy, bilateral splanchnicectomy (subabdominal and complete) and thoracoabdominal sympathectomy are very confusing

If relief of chronic pain and digestive disability is obtained following paravertebral block (chapter 10), equally good results may follow splanchnicectomy Similarly biliary tract symptoms which are relieved by vagu *blocking agents may also be relieved by vagotomy*

Grimson describes a celiac ganglionectomy which can be done through a right upper abdominal *incision* The ganglia on the right are exposed through the gastrohepatic omentum, are mobilized to the right and excised On the left it is necessary to incise the base of the transverse mesocolon to the left of the duodenal jejunal junction, dissecting above the left renal vein in the retroperitoneal space Branches to the adrenal are usually severed by this procedure

Celiac ganglionectomy has been variously recommended as most efficient *in abolishing abnormal spasm at the papilla of Vater It would appear* to be less mutilating and more efficient than thoracic splanchnicectomy *and thoracic sympathectomy*

Unfortunately these denervation procedures do not apply solely to the biliary tract Other intestinal innervation is also altered Local denervation of the bile ducts cannot be fully satisfactory by reason of the numerous nerve plexuses which accompany all vessels

Peptic ulcer perforation or severe pancreatic *necrosis have been reported* as occurring following both splanchnicectomy and vagectomy These *had not been recognized in the absence of afferent pain or similar impulses*

Splanchnicectomy sympathectomy and vagectomy should not be accomplished at the time of primary operation on the biliary ducts There

should be adequate study consultation and evaluation available prior to electing a denervation procedure as definitive therapy

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15

POSTOPERATIVE STATES

A The Uncomplicated Postoperative Period

Rehabilitation of the patient starts at the time of operation on the biliary tract Proper position adequate anesthesia and fluid management with plausible physiologic and anatomical surgical procedures will permit rapid recovery to normal states Operative manipulations and postanesthesia reactions usually prohibit oral alimentation for 24 hours In this interval blood loss is replaced fluid requirements are met intravenously and the patient is assisted in return to homeostasis

Blood transfusions are given at operation to replace blood volume only after loss has occurred Particular care is exercised that blood not be given to excess in patients with hypertension myocardial damage pneumonitis and hemoconcentration due to excessive fluid loss

Fluid requirements vary with environmental temperature and the individual's specific loss 1000 to 2500 cc of glucose in water and saline are usually required in the 24 hours postoperatively It is not usual for the patient following biliary tract operations (except those with pancreato duodenal resection) to require parenteral alimentation beyond 36 to 48 hours

However during the first 12 to 18 postoperative hours oral intake is discouraged If nausea and vomiting continue after 8 to 12 hours gastric lavage is required The nasogastric tube may be left in only to overcome gastric dilatation Intermittent aspiration of the stomach is very efficacious As soon as water is retained liquids and a bland diet are immediately started The patient's desires for specific foods are encouraged High carbohydrate liquids are offerred constantly By the third day the patient is usually eating and enjoying a moderate diet Vitamin C K or B may be administered

Laxation may be required Milk of magnesia mineral oil prune juice cereals and bland bulk stimuli depending upon the patient's preoperative bowel habits are usually reliable Glycerin suppositories and enemas may be helpful after the second or third postoperative day

Urinary problems are usually minimal There is usually no contraindication to sitting being out of bed or walking as soon as the patient has satisfactorily overcome the immediate operative and anesthesia effects

Early ambulation prevents many genito urinary peripheral vascular and pulmonary sequelae Even in patients whose myocardial reserve is made quate activity is valuable All patients move all limbs and joints several times daily under medical observation Particular attention is devoted to specific respiratory movements of the right lower chest An abdominal binder is helpful provided that it does not restrict costal excursions

Oral and hypodermic analgesia is essential to comfort

Oxygen by catheter or tent is given to any patient who has a doubtful or deficient myocardial reserve until blood pressure and pulse rate have positively stabilized Should high concentrations of oxygen discourage adequate respiratory movements it may be discontinued

Antibiotics are not essential in patients who do not have infection Benefit derived in one patient which prevents the occurrence of a naso pharyngitis is more than offset by the serious after effects of hypersensitivity to an antibiotic Should one be required by virtue of an active cholecystitis or phlebitis or because gastrointestinal flora are to be discouraged a broad spectrum antibiotic should be selected for use preoperatively The organisms which are encountered as in empyema or cholangitis should be identified regarding sensitivities to antibiotics

Removal of drains and sutures is the personal prerogative of the operating surgeon The patient may be discharged to his home when he is able to care for his personal requirements in the bathroom when he can feed himself and can walk short distances Prolonged hospitalization should not be required except for complicated problems

When discharged the patient is given five instructions

1 Hair can be shampooed Sponge bath is taken until incisional area is clean Tub or shower (preferred) may be then taken provided someone assists patient in and out of tub to avoid accidental falls
2 The patient wears an abdominal *(elastic) support if a large overhang* is present or if there be an incisional weakness Garment usually is not required after six weeks
3 Patient is permitted to ride in a car and to walk increasing distances Stairs may be climbed but one flight at a time and the patient rests between ascent and descent The patient is instructed to nap several times daily There is to be no push pull or lift done against resistance
4 For several months chocolate spices cabbage beans excessive fat and butter are avoided Small amounts of butter and oleomargarine are used to increase palatability of food Restricted foods may be started gradually when the patient is fully recovered
5 The patient is instructed to report to his physician and follow his physician's regimen A date is given for follow up examination in six months

B Emergencies during the Postoperative Period

1 *Surgical Problems*

Early reoperation has been discussed (chapter 14) with reference to hemorrhage, peritonitis, increasing jaundice and intestinal obstruction

Hemorrhage requires adequate blood replacement, sedation and prevention of anoxia Careful observation of the blood pressure, pulse rate hemoglobin and red blood cell levels are collateral evidence to the extent of bleeding Increasing abdominal distention hematemesis and pallor, thirst and apprehension are systemic signs of continuing uncontrolled blood loss When these appear within four to six hours of surgery and do not abate subsequent to blood transfusion, *reoperation is frequently required* Administration of vitamin K intravenously (indicated for hypoprothrombinemia), ascorbic acid (for specific deficiency) and toluidine blue or protamine (as non specific agents) ocassionally have been efficacious in controlling liver bed oozing which is a frequent cause for postcholecystectomy bleeding Intravenous albumen and fibrinogen are indicated in certain bleeding states Increased portal hypertension may follow biliary tract procedures and can augment gastrointestinal bleeding

After 36 to 72 hours bile drainage may follow simple cholecystectomy There may be additional leakage around an indwelling "T" drain or tube in the patient who has had a choledochostomy Occasionally, this is accompanied by increasing abdominal distention and high fever and the patient becomes *lethargic flushed and perhaps irrational Increase in bile* drainage may indicate that a duct ligature has slipped, that an anomalous duct has been cut or there is some unforeseen intracholedochal abnormality Unless symptoms of peritonitis subside within 18 to 24 hours more adequate external drainage of bile is required Should the patient's condition permit definitive repair or revision is done *A bile fistula is not fatal per se*

Icterus may appear following any operation on the bile ducts Although duct obstruction is an obvious cause other factors may be present Careful differentiation among hemolytic, traumatic and organic causes may take several days Repeated examination of urine, stool and blood for bilirubin and urobilinogen levels is essential Increasing bilirubinemia and absent urobilinuria are evidence of organic obstruction Antispasmodics such as *nitrites or papaverine may be given and* magnesium sulfate introduced into the duodenum Unless bile flow appears in the intestinal tract within several days an organic or traumatic cause for bile duct obstruction probably exists and its relief is indicated Drainage of the bile ducts *alone* may be done Should the patient's *condition permit*, definitive repair and *reconstruction may be indicated (chapter 12)* This is not urgent

Ileus gastric dilatation and intestinal obstruction can be important

problems It may be necessary to establish an enteroenterostomy between loops of an enterocholecystostomy to relieve an internal hernia through a defect in the mesocolon or to relieve obstruction due to the rapid formation of intraperitoneal adhesions

Other indications for urgent reoperation do not particularly appertain to the biliary tract These include ligation of femoral veins in selected patients with phlebothrombosis and emergency tracheotomy in patients with respiratory obstruction or inefficiency

2 Medical Problems

Atelectasis is most common in the postoperative patient Encouragement of full respiratory movements and an adequate cough will prevent this to a great degree Sharp slaps on the back at the level of the third or fourth thoracic vertebrae may inaugurate an effective cough Aspiration with a catheter or through the bronchoscope may be required Potassium iodide therapy or medicated steam inhalations (using *croup tent*) are valuable in assisting expectoration of sputum

Respiratory depression may be due to idiosyncrasy hypersensitivity or overdosage of drugs In respiratory depression secondary to barbiturates picrotoxin or Metrazol are carefully administered Likewise Nalline is used to counteract unwelcome effects of opiates

Antibiotics for prophylaxis against respiratory infections in cases of respiratory depression are indicated As a result many patients develop immediate or delayed reaction to them Small doses of epinephrine adequate dosage with aminophylline use of the antihistamines and occasional administration of Cortisone or Corticotropin will usually relieve an acute state of hypersensitivity The usefulness of adrenal cortical extract has not been sufficiently emphasized However it should be pointed out that Cortisone and Corticotropin tend to retain salt and water (increasing edema and ascites) and may also be responsible for the development of portal or splenic vein thrombosis Particular attention is necessary to prohibit adrenal cortical deficiency which may occur (and be fatal) in patients who had previously been given courses of therapy with Cortisone or ACTH Use of cortisone in adequate dosage is required both pre and postoperatively

When and if phlebothrombosis develops conservative measures are best Supportive bandages rest with selective activity and intermuscular trypsin (parenzyme) are used Anticoagulants are not contraindicated even as early as three days postoperatively It is suggested however that heparin administration be very cautious

Congestive heart failure and coronary occlusion require symptomatic therapy With a failing myocardium digitalis and mercurials are used

Quinidine procaine amide and Mecholyl are indicated in treatment of various arrhythmias

Anuria may appear postoperatively as a manifestation of prolonged hypotension, as secondary to an incompatible blood transfusion and from other causes Management (chapter 6) depends upon replacement of fluid and blood deficits without overloading hepatic, cardiac or renal function

Hyperpyrexia may occur as a startling development in the patient who has been jaundiced or emaciated Liver cell glycogen is depleted and anoxia is superimposed upon failure of normal hepatic and renal mechanisms Theories concerning its cause relate to sepsis secondary to anaerobic infection of the liver, effect on the mid brain and specific failure of a thermo regulating enzyme produced by the liver Its appearance is not always of bad prognosis It may be relieved following adequate administration of glucose blood and protein antibiotics and sedation An electrically regulated cooling blanket is valuable, i e Thermorite machine

C Evaluation of the Bile Ducts Postoperatively

The state of the common bile duct after choledochostomy is evaluated in several ways First, the clinical course is observed after the external limb of the "T" tube has been clamped Second the duct is studied by cholangiogram Third, measurements are made of intracholedochal pressures and volumes including a study of the response to added measured fluid

There are defects in each of these methods The best upon which we rely greatly is the cholangiogram However in some instances the radiologist may not be able to give an unqualified opinion without repeating the study Even then the conclusion may be doubtful In such instances and in fact in all cases it is desirable to employ clinical tests to confirm and to supplement the cholangiogram

Postoperatively the daily volume of bile drainage from the "T" tube is measured As this decreases to less than 250 cc, usually after the fifth day the external limb of the 'T' tube is clamped for increasing periods At the end of one of these periods a three way stopcock is attached to the external opening of the 'T' tube A 20 cc syringe and a water manometer are also attached to the stopcock

Pressure in the common duct is measured by reading the fluid level in the manometer The contents of the duct are aspirated Normal saline solution or 1 per cent procaine hydrochloride is then injected into the duct Usually from 10 to 30 cc of fluid is injected during three to five minutes Measurements are recorded at the conclusion of the injection or at the first sign of abdominal discomfort Pressure levels are observed for five to ten minutes or until the reading is zero depending upon the rate of duct

emptying Changes in pressure and volume of the biliary tract are recorded The impression gained concerning the duct is charted as patent "partially obstructed" or completely obstructed or an individual description is given

The residual volume of the postoperative common bile duct is normally 5 to 6 cc This varies because of variable base line factors such as food stimuli state of duodenal tonus volume content of the duodenum intraperitoneal pressure and the patient s state of mind Residual volume greater than 12 cc is usually associated with some degree of obstruction at the termination of the common bile duct

The normal pressure in the common bile duct during the resting state may range up to 7 cm of water When pressure is increased over 10 cm obstruction of some character to bile flow is present

As fluid is *injected into the duct there is a rise in pressure until 15 to* 18 cm of water pressure is present after the injection of 10 to 15 cc of fluid At this level there may be moderate discomfort and a sense of abdominal fullness This is relieved spontaneously as the intracholedochal pressure is decreased

The duct normally empties its contents into the duodenum at a rate of 2 to 5 cc per minute

In patients with choledocholithiasis four pictures may be seen

1 If the life history of the stone is short the common duct volume is less than 15 cc although the maximal pressure may rise to 25 to 30 cm This duct empties slowly or may not empty
2 When the common duct stone has existed for many months the volume of the duct is usually over 15 cc whereas the maximal intracholedochal pressure may be less than 20 to 25 cm This duct empties slowly and painfully
3 The duct which once contained a calculus has a volume greater than 10 to 15 cc with a pressure lower than 10 to 12 cm This duct empties very rapidly (10 to 11 cc per minute)
4 A retained stone in the hepatic ducts may not present any abnormalities on pressure volume study

In patients who have edema of the papilla redundant mucosa or spasm of the papilla after passage of probes and dilators at operation and in some patients with pancreatitis a bizarre picture may be seen Pressure within the duct may be low the rate of flow slow and the pain threshold low (10 to 12 cm of water)

In patients who have been demonstrated to have reflux into the pancreatic duct no significant alterations have ever been observed Only 10 per cent of these have pancreatitis

In the measurement of intracholedochal pressures and volumes there

are a few precautions to be observed The 'T' tube placed poorly at operation may cause irregular flow in and out of the duct The closure of the duct around the tube may be defective, permitting leakage There may be a kink in the transabdominal course of the 'T' tube There may be a very short "T" tube

The hepatic ducts' content may vary from 3 to 5 cc and in some cases these ducts may not fill In an occasional patient the cool temperature of the injected solution may provoke duct spasm particularly when the papilla already has an altered threshold of relaxation

In addition the zero point of the manometer must be placed at the level of the common duct We place the zero point at the middle of the sagittal plane of the body with the patient recumbent

Pain reactions are to be evaluated with regard to distensibility of the common duct wall, irritative reaction at the papilla pylorospasm and other conditions

D Late Sequelae of Bile Duct Surgery

1 Dyskinesia

Irregular emptying of the bile ducts may be the basis for symptoms These may precede or follow surgical procedure concerning the bile ducts Such hypertonic and hypotonic abnormalities associated with pain flatulence and indigestion have been identified as biliary dyskinesia

a Hypertonic

The hypertonic type of biliary tract dyskinesia is seen when the bile passages (and gallbladder) exert a greater fluid pressure than normal when pressure waves are more frequent and when fluid absorption from the duct is normal or less The usual cause for increased intraductal pressure is an obstructive mechanism at the papilla In the absence of organic disease or foreign substances the block to flow can be due to dysfunction within the muscle mass at the papilla

Infection of the ductal or duodenal mucosa at the papilla a calculus or extrinsic disease may alter normal reactivity at the papilla Higher pressures within the duct may be required to permit the sphincter to relax Continuation of the irritation may establish a vicious cycle wherein the increased sphincter resistance prohibits normal drainage of bile which establishes further noxious stimuli These stimuli may result in fibrosis at the sphincter superficial ulcerations particularly in the duct valvules, infections within ductal glands diverticula and fistulae

Intermittent sphincter resistance may be identified as spasm if the duct dilatation is minimal and if biliary tract function is otherwise normal

This hypertonic sphincter mechanism may continue and increase A stricture will not result from abnormal activity but follows trauma or infection

Hypertonic biliary dyskinesia is characterized by an increased intraductal pressure with or without an increased intraductal volume It is usually seen with calculous choledochitis after passage of dilators and sounds and in the presence of organic obstruction Relief of the obstructing mechanism is usually obtained by sphincterotomy or papillotomy

b Hypotonic

Hypotonic biliary dyskinesia depends upon the inability of liver gall bladder and bile passages to provide intraductal fluid pressures sufficient to stimulate the sphincter at the papilla to open Slow excretion and formation of bile by the liver may be augmented by rapid absorptive rates of fluid within the gallbladder or by a great capacity for the ducts to expand Characteristically bile in patients with hypotonic dyskinesia is thick and viscid Painful episodes occur as frequently as abnormal distention appears Denervation procedures do not guarantee that distention will not occur but merely obviate its recognition These are indicated only under such conditions wherein free unobstructed and continuous flow of bile is guaranteed in response to normal digestive stimuli The free flow of bile through an ample conduit should be present at the conclusion of any surgery involving the bile duct

2 Postcholecystectomy Syndrome

Irregularities in bile duct function frequently follow cholecystectomy they may have been instigated by the same mechanisms as produced cholecystitis and lithiasis Cholecystectomy for chronic calculous cholecystitis may be expected to bring relief of symptoms to 95 per cent of patients When complicating or additional disease exists at the time of cholecystectomy full relief of symptoms may not be expected Statistics regarding the incidence of symptoms following cholecystectomy are difficult to evaluate (4 to 40 per cent)

The postcholecystectomy patient may have an occasional incisional discomfort rarely associated with digestive symptoms and continued inability to digest certain foods adequately On the other hand the patient after cholecystectomy may be deeply jaundiced with sepsis diarrhea and vomiting Both states are within the postcholecystectomy syndrome

Common duct function in the patient who has a non functioning gall bladder is not greatly affected by cholecystectomy since all biliary tract function has already been transferred to the common duct However a gallbladder with cholelithiasis can still function as a surge chamber to retain and concentrate bile for digestive purpose and respond normally to

digestive stimuli When a functioning gallbladder is removed readjustment in function by the common bile duct may be delayed In most patients, within one to three weeks full and complete asymptomatic transport of bile through the duct will occur In those patients who have an inadequate conversion of the common bile duct to asymptomatic bile transport symptoms of dyskinesia may appear

Failure of the common duct and papilla to be coordinated then continues as "postcholecystectomy syndrome" Intravenous cholangiography is indicated for diagnosis (chapter 8)

Many other conditions have been identified as this state Voluminous reports in the literature have appeared to present surgical trauma and reflexes from other organic disease among etiologic factors Functional dyskinesia of the common bile duct should be differentiated from those which have been *(a) overlooked at previous surgery*, *(b) produced by* previous surgery (c) produced by other disease (d) symptoms due to *another ailment*

Incomplete or hasty maneuvers during cholecystectomy may overlook *calculi in the common bile duct These are the most common cause for* persistent or recurrent postcholecystectomy symptoms Incomplete di*section may permit a long stump of cystic duct to remain often parallel* to the common bile duct This stump may have calculi form in it Occasionally incomplete dissection may *have been hindered by an anomalous* vessel which also masks anomalies such as a reduplication of the common *bile duct Tumor of the posterior wall of the stomach pancreas or papilla* may be present together with cholelithiasis and be clinically silent Tumor *elsewhere as in the kidney and colon may also be missed at the time of* cholecystectomy Pancreatic heterotopia is usually not responsible for a *missed diagnosis but is confused with malignancy In addition, cholangitis* may be present at the time of cholecystectomy and unless the common duct *is drained adequately these patients do not return to good health*

There are several traumatic features of cholecystectomy which are *followed by symptoms Those of stricture and fistula are only too obvious* Those in which a neuroma is formed or bile leakage into the gallbladder *bed forms a pseudocholedochal cyst are difficult to identify* Surgical errors in commission form the fewest causes for the postcholecystectomy syndrome

Pre existing or concomitant disease is responsible for much of the symp*tomatology following cholecystectomy* Errors of omission failure to identify additional disease or inability to observe an obscure or hidden *lesion is the most common cause for persistent discomfort after removal of* the gallbladder Such co-existant disease include peptic (duodenal) ulcer

and hyperacidity, pancreatitis and pancreatic cyst hepatitis and cholangiolitis esophageal and hiatal hernia and allergic states

Careful clinical differentiation is difficult in many cases However gastric duodenal and pancreatic lesions are often associated with biliary tract dysfunction and pseudobiliary colic even after cholecystectomy Similarly associated choledolithiasis cannot be expected to be cured even though gastroduodenal or pancreatic abnormalities are corrected

The patient who has skeletal disease including metastasis or who has a psychoneurosis should not be subjected to cholecystectomy for relief of those symptoms Similarly symptoms will persist after cholecystectomy if they were due primarily to intercostal neuralgia localized angioneurotic edema (Quincke) intra abdominal adhesions intra abdominal hernia chemical indigestion colitis or calculous pyelonephritis

3 Residual Choledocholithiasis

Choledocholithiasis usually occurs following migration of gallbladder calculi Such stones are rarely greater than 0 5 cm in diameter at the time of passage through the cystic duct They can enlarge

Such stones in the common bile duct may give no hint of their presence at the time of cholecystectomy Classical history may be absent and dilatation or thickening of the common bile duct may not be obvious at operation Under certain circumstances calculi may not even be visualized by operative cholangiography through the cystic duct

Even when the common bile duct has been opened and calculi removed it is often difficult to establish that the biliary tract is free of residual calculi (chapter 12) Adequate drainage through the papilla must be assured in such cases Residual calculi may be hidden behind a duct valvule in a diverticulum or high in an intrahepatic duct These may then appear postoperatively although the patient can be symptom free for months or years

Certain individuals have a predisposition to manufacture calculi In such cases long term drainage is used to reinforce an adequate stoma at the termination of the bile duct

Intrahepatic calculi do not produce a specific clinical picture They may cause recurrent colic Their presence may be suspected at operation if the extrahepatic ducts contain sand small stones or inspissated biliary pigments At surgery a segment of the liver may be occluded as visualized by cholangiogram and there may be segmental bile staining or cirrhotic changes

The duration and character of colic following cholecystectomy may suggest residual choledocholithiasis If colic recurs within a short time

after cholecystectomy a 'missed' stone may be suspected Colic may not appear until years after cholecystectomy, accompanied by obstructive jaundice, chills and fever, and also be due to a residual stone

When bile duct obstructive symptoms appear following cholecystectomy, drainage of the bile ducts is imperative Removal of obstructing mechanism is indicated but may not always be feasible

Residual stones are frequently seen on postoperative cholangiographs Up to 8 to 10 per cent of patients have been reported with additional stones in the duct following choledocholithotomy

Opportunity for study of recurrent biliary tract lithiasis is limited Several Swedish surgeons followed over 50 patients for more than 30 years after cholecystolithotomy Recurrent stone formation in the gallbladder was observed in 31 per cent Actual formation of new calculi in the bile ducts has been suspected in all cases of residual choledocholithiasis but proven in only few

Reoperation for removal of residual calculi is not necessarily a formidable procedure particularly if a tube is retained within the bile duct (chapter 12) However there are personal medical and surgical contraindications to reoperation Under such circumstances various conservative methods for therapy have been recommended These include the use of solvents irrigation flushes and intraductal manipulation

Many reports have appeared concerning the fragmentation and dissolution of stones in the common bile duct by chemical means through an indwelling tube Pribram has reported many cases in whom he used ether and mineral oil for periods up to six weeks Walters and Wesson and Burgess have supplemented the use of ether oil with nitrites Best utilizes choleretics in addition to chemical solvents and pharmacologic relaxants through a specially constructed T tube Other investigators have successfully used chloroform solution G nupercaine varidase hyaluronidase tryptar and combinations of the above It is valuable to obtain a gallstone to test solubility in the proposed reagents by 'in vitro' contact I have seen several instances where ether irrigations were continued for six to ten months to be finally successful

If the lumen of the 'T' drain is wide (22 Fr) a flexible foreign body forceps may be introduced into the duct directly or through an infant's cystoscope Direct observation is possible through the 'T' tube with the cystoscope Better results are obtained by utilizing radio opaque dyes and fluoroscopic manipulation with the *flexible lithotrite* In two instances I have been able to fragment a calculus by this maneuver The fragments were later irrigated from the duct by *copious quantities of saline*

A double lumen 'T' tube permits introduction of solution through one lumen and return flow through the *other* This is very valuable in patients

with residual choledocholithiasis Direct irrigation is supplemented by choleretics Among these are magnesium sulfate and Decholin Decholin's effect is to dilute bile and increase the amount of fluid volume in the bile and bile acids without increasing the concentration of bile pigment and cholesterol The diluted bile in larger volumes tends to increase intra choledochal pressure and to force material through the papilla if the papilla maintains its normal sphincter tone Magnesium sulfate does this except that it permits relaxation of the sphincter If the sphincter is open at the time a large volume of bile is flowing then it is feasible that fragments of calculi 2 to 4 mm in diameter would be passed through normal channels

Dr Best of Nebraska recommends the following procedure for management of residual stones Three Decholin with belladonna tablets are taken daily after three meals and at bedtime for three days Each morning before breakfast the patient takes six ounces of magnesium citrate or phosphate soda plus three or four tablespoonfuls of cream or olive oil before noon and evening meals Whenever there is extreme discomfort and after the evening meal nitroglycerin ($^1\!/_{100}$ gr or $^1\!/_{200}$ gr) is given sublingually The duct is irrigated daily with warm saline Three to 5 cc of chloroform or ether is then introduced through the T tube Flow may be directed towards the lower end of the common duct or towards the liver in certain types of T tube Two grains of papaverine (hypodermic) or sublingual nitroglycerin can be given following this

If choledochitis cholangitis or hepatitis appear or if the common bile duct increases in size conservative measures should be discontinued The stone should be removed surgically

There is available an operative method for removal of intracholedochal calculi based on preliminary work by Dr Dees of West Virginia (Chapter 11)

4 Problems Following Pancreatoduodenectomy

In addition to the technical hazards of pancreatoduodenectomy there are the demands of postoperative management Problems of bile and intestinal fistulae of vomiting and parenteral nutrition are to be met In addition there is required a maximal effort towards stabilization of body economy (homeostasis) due to withdrawal of pancreatic function

Effect of internal and external enzyme loss is increasingly proportional to the amount of pancreatic tissue which is removed Transient appearance of effects of total pancreatectomy may appear in subtotal resection There are three major physiological consequences of total pancreatectomy (a) altered glucose metabolism (b) altered digestion and absorption of fats and proteins (c) irregularities in lipid metabolism

Resection of the pancreas is an invitation to diabetes However, these patients do not require as much *insulin as the spontaneous diabetic* They may need 20 to 30 units of insulin daily Larger amounts of insulin have been fatal *Continuous observation is mandatory* In addition there is a close correlation between glucose and fat metabolism It has been demonstrated *that alterations in the enzyme systems for fat synthesis play* a substantial part in the diabetic and following pancreatoduodenectomy

Aberrations in fat and protein digestion are rapidly manifest following pancreatectomy by appearance of a gray, bulky, soft and diarrheic stool Intravenous fat emulsions are intermittently beneficial The patient after pancreatectomy is not known to become obese

Lipid metabolism is still not fully appreciated The beneficial use of lipotropic drugs or amino acids *such as choline and methionine has not* been proven Certain unexplained difficulties in lipid metabolism undoubtedly *affect nutrition following pancreatectomy*

Protein and vitamin metabolism depend to a large extent upon adequate *hepatic function Pancreatic gland effect on the liver cell has not yet been* fully investigated although studies of plasma proteins indicate that certain enzyme systems are damaged by pancreatic disease

Deficiencies in internal metabolism are greatly *magnified by loss from* vomiting and diarrhea and from bile and intestinal fistulae

Sodium, potassium and chloride may be lost by suction from the gastrointestinal tract In vomiting more chloride is lost than sodium On the other hand diarrhea leads to greater depletion of sodium and potassium than of chloride Analysis of the materials lost by these routes is impractical *However there are solutions available for intravenous* feeding of more base than of chloride or vice versa which provide adequate replacement of *losses (chapter 7)*

Hypochloremic alkalosis develops whenever there is intracellular potassium depletion resulting *from bile loss and from other disturbances in* electrolyte balance When alkalosis reflects a low cellular potassium, the administration of sodium chloride will aggravate symptoms by further depletion of the potassium as the sodium becomes the intercellular substitute for it Accordingly under such circumstances adequate intravenous *potassium is indicated to supplement* other agents used for parenteral alimentation

Hemorrhage, shock and *severe infection* further modify the problems of fluid and electrolyte balance In the cirrhotic patient particularly there is the additional problem of hypoalbuminemia associated with intraperitoneal and renal loss of plasma proteins Use of intravenous serum albumin

is valuable Administration of gamma globulin may be very useful when deficiency exists

Bile loss alone as through a permanent fistula frequently produces hyperchloremic alkalosis usually associated with the effect of potassium depletion Bile may be fed or replaced in toto through a jejunostomy tube

On the other hand, concomitant renal disease can produce an hypochloremic acidosis For example W C (aged 67) had pancreatoduodenal resection for carcinoma of the pancreas at the papilla of Vater Eighty per cent pancreatectomy was done the pancreas implanted to the duodenum by end-to-end anastomosis The patient had severe renal insufficiency in addition to jaundice Postoperatively a bile fistula lost 300 to 400 cc daily He was unable to retain foods and vomited unless nasogastric tube suction was employed In addition there was excessive perspiration due to hot weather Although glycogenolysis was adequate acetonuria was present Oliguria of less than 200 cc daily appeared on the fifth postoperative day Chemical examinations revealed a urea nitrogen of 100 mg % and a hypochloremic acidosis This patient was given intravenous sodium lactate calcium gluconate and blood transfusions in limited quantity to approximate his fluid loss Three days later renal output and chemical determinations were normal Potassium and proteins had been normal all the while Oral feedings were then retained, the bile fistula closed spontaneously The patient is well

Cortisone and Corticotropin may have a disadvantageous effect in such patients because of the tendency to produce water and salt retention However there is often seen a temporary stimulation to appetite and improvement in attitude Its effect on wound healing and general status following pancreatic resections has not been reported

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